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THE PSYCHOLOGICAL APPROACH TO PAIN*

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Durban

I have been asked as a psychiatrist to confine my remarks in this symposium* to the psychological approach to pain. It will be appreciated, however, that psychiatry is rooted in genetics and physiology and demands not only a knowledge of psychology but some acquaintance with sociology, anthropology, history and philosophy.

It is true that the clinical approach of the psychiatrist is similar to that of the physician. For both the essentials of diagnosis are the same—the site of the disease, its causes, and the nature of the functional disturbance. But the psychiatrist begins his investigation of causes and his search for their quantitative relationship on almost virgin soil. For our clinical purposes, it is agreed that people may become ill for psychological or physical reasons, or both; where we might differ is in the emphasis we place upon one or the other, psychological or physical.

Those concerned merely with measurement and the classification of sense data might offer a purely physiological hypothesis of mental symptoms. Others might continue to regard mind as a 'thing in itself', separate and distinct from any physiological emphasis. Men like McInnes[2] have posed an important question, whether the psychic life is a reality which would always require its own symbol 'mental', or whether one day this concept, too, will be explained and understood in terms of physiology. I think, however, that today we all accept the idea that a person is an integrated whole, of which mind and body are distinguishable but inseparable aspects. It is always difficult to avoid language which suggests dualism, and it is obviously convenient to think in terms of this dualism, provided we appreciate that it is 'the person as a whole' who is an entity, and that body and mind are two aspects of this whole person.

Russell Brain[3] has said that in normal persons, the unconscious motivation of conscious acts were not necessarily to be explained in terms of physiology, or by the presence of an affective disorder. A neurosis is a disorder of values, and it is not always possible to call in a cerebral disorder to explain it. Trends in modern research suggest that emotional states are psycho-physical conditions arising in the organism as a result of some failure in adaptation. They express themselves both in objective physical signs and subjective mental symptoms. These physical signs and subjective mental symptoms, as Miller[4] has reminded us, constitute a unitary experience which may be the result of a cortical disturbance, an alarm reaction from a particular organ, or an upset of normal body chemistry.

To understand the psychology of pain, it is essential, I think, to appreciate what I have referred to as this 'unitary experience', and that it is 'the person as a whole' who is the entity.

PSYCHO-PHYSICAL CONSIDERATIONS

We have been taught that the sensation of pain arises as a result of the stimulation of certain nerve endings and that the impulses set up are transmitted through the afferent paths of the nervous system to the thalamus, where they are appreciated in consciousness. But by dividing the thalamo-frontal tract a personal pain may be reduced to an experience not unlike the sympathetic appreciation of pain in others, or as a patient once described the change following a leucotomy, 'Oh, yes, doctor, I still have pain, but it is no longer my pain'.

One would imagine from a purely physiological basis that so long as the nervous pathways were intact, the sensation of pain would always be established and persist. We know, however, that this is not always so, for under hypnosis or in hysteria the sensation of pain may be completely absent. This at once suggests the appearance and existence of other factors governing pain sensation, factors concerned with that other part of the person as a whole which we call 'mind'.

Pain may be produced by damage or excessive heat or cold to the skin, but in the internal organs these stimuli do not produce pain. In these tissues the effective stimulus is tension, either from distention, contraction or anoxia. Pain may also arise from inflammation or irritation of the afferent nerves themselves. Now it is common knowledge that memory is reinforced by repetition and emotion, which

* A paper read in a symposium on the Treatment of Pain (opened by Sir Russell Brain, Bt.[1]) at the South African Medical Congress, Durban, September 1957.

may explain why a pain may continue after the original stimulus has ceased. Pavlov, for instance, suggested that the excitability of the cerebral cortex may be affected by emotion. Pain may also be conditioned like conditioned reflexes. And closely related to this is the emotion of fear, which is again associated with any kind of threat to security.

The treatment of a painful tendon by the injection of a local anaesthetic, which only lasts for a short time, may often cure the pain permanently, the cure probably depending on the elimination of some conditioned reflex and the realization by the patient that he can now move the part painlessly while he does not know how long the anaesthetic will last.

I mentioned above the reduction of pain occurring in hysteria, when a patient may become quite insensitive to injury. It is also reduced by suppression, by persons of strong will, and it is a common experience to find pain alleviated when the patient's attention is diverted, just as fear is relieved by giving a patient something to do. This suggests that stimuli from outside take priority in consciousness over minor bodily sensations. In passing, it is worth noting how important is the visiting of the sick, in diverting their attention.

Then again, pain may be increased by anxiety. Normal bodily sensations may be mistaken for something more serious; a condition common in athletes, who are particularly body-conscious. It is sometimes difficult to distinguish between an anxiety state and hysteria, since by claiming pain the patient may escape having to perform an unpleasant duty. The fact that pain can be relieved by psychological means does not always prove that it is neurotic, for we know that organic pain can be relieved by hypnosis.

It seems clear, then, that the extent to which certain impulses give rise to pain depends on the personality of the patient. Personality, in fact, often dictates the sensation and its intensity. A given injury may produce pain out of all proportion to that experienced by the average person or, conversely, it might not produce the pain that would normally be expected.

PERSONALITY

The statement that the degree of pain experienced by a patient depends on his personality as well as the nature of trauma or stress, requires a definition of personality. McDowall has described it as 'the resultant of the total forces concerned with the formation of mind, the heredity, together with the personal experience of the individual as shown by his relations to his fellows and their institutions'.

This definition, acceptable to some psychologists, does not seem satisfactory for our purpose. It excludes the pathological conditions, such as anxiety states and hysteria, to which I have referred. As Mayer Gross[5] has pointed out, 'However far the rapprochement goes, between psychiatry and psychology, we still cannot expect that psychiatry will ever be able to base itself on psychology alone. The psychiatrist can never lose his interest in what is happening in the bodily field, and he will seek wherever he can the direct contacts with physiology and medicine. Furthermore, the primary concern of the psychiatrist is with the morbid mental states and, however much is known about the psychology of the normal individual, in the pathological field new laws will be found to operate'.

PSYCHOPATHY AND NEUROTIC REACTIONS

I have said that the approach of the psychiatrist is similar to that of the physician. For both of them the 3 essentials of diagnosis are: the site of the disease, its causes, and the nature of the functional disturbance. It therefore becomes necessary to consider briefly the aetiology of psychopathy and the neurotic reactions.

During the first World War psychiatric attention was mostly concerned with neurotic symptoms (shell shock), and on its psychological causes. In the second World War, however, much more attention was given to the constitutional background. The constitutional background could be measured to some extent by taking a careful history, and constitutional mental instability was indicated by a family history of mental illness, previous nervous break-down and an unsatisfactory work record. It was found that the liability to nervous break-down varied with the amount of stress, but also with the constitutional make-up of the individual. Further, it was observed that some men were vulnerable to particular forms of stress, which varied with their temperamental make-up and their previous experience.

These reactions should not, of course, be regarded as of equal importance. The cold and callous personality, the constitutionally neurasthenic personality, these indicate a serious disorder of structure. On the other hand, hysterical conversion syndromes or acute anxiety states, caused by situations of sudden stress, cannot be so regarded.

DIAGNOSIS

In the absence of clinical signs of organic disease, the diagnosis of the cause of pain is one of the more difficult problems in general practice.

Investigation should consist of a careful biographical history and a meticulous physical examination. The symptoms should then be assessed in their complete setting, their longitudinal section, their various cross sections and the circumstances in which they occur.

This biographical history should include a careful consideration of the heredity (family investigations) and somatic, psychological and pathological factors, in an attempt to distinguish symptoms that are part of a personality problem from those that are impersonal and organic.

For example, the patient with a hysterical personality describes his symptoms in superlatives, and it is often impossible to determine the exact site of the pain, or even what he does feel. In organic pain, the patient may be preoccupied with his symptoms, but the history-taking and the physical examination is an obvious added burden. The pain is seldom continuous, and he freely admits periods of relief. He looks ill, and has often lost weight from prolonged suffering.

The differential diagnosis from a state of depression may sometimes be difficult, but must be attempted because of the possibility of treatment; in primary affective disorder by convulsive therapy, in chronic neurasthenia depending on the estimation of the basic personality, the extent to which the environmental stress has contributed to the condition and

the extent to which these stresses can be avoided in the future.

It must not be forgotten that a patient may develop a cerebral tumour even though he has a hysterical personality. I well remember such a patient being transferred from the general wards of a Royal Naval Hospital during the war as a 'malingerer complaining of backache' to the neuropsychiatric department, where a diagnosis of spinal tumour was soon established.

The tendency to classify human beings into types is an over-simplification of the problem, however useful it may be in practice. There is statistical evidence to show that a high correlation exists between physique and temperament, which suggests that the physiological and psychological attributes of the individual are aspects of an underlying unity, and therefore aspects of one another.

I have pointed out elsewhere[6] that fixed and rigid personalities tend to develop single symptoms, while immature and dependant types develop multiple symptoms. It is also important to remember that in recurrent hypochondrical attacks with obsessional characteristics the underlying depression often remains undetected, and there is then a grave risk of overlooking the possibility of suicide.

TREATMENT

In considering the psychiatric treatment of pain there is no need for me to dwell on what is already familiar—the physical examination, the psychiatric interviews, and the routine psychiatric methods; but I should like to stress the increasing importance of the electro-encephalogram in personality assessment.

Lennox[7] and others have shown that brain waves vary with the physiological state of the body, i.e. in hyperventilation, lowering of the blood sugar and overhydration. The brain waves of the normal adult differ from those of a child. In normal development the patterns of childhood gradually give way to those of the adult as the psyche develops. The presence in the adult of brain-waves like those of the child is abnormal, and is associated with emotional immaturity and constitutional instability of nervous control.

The clinical relationship between physician and patient, like all other human relationships, will always remain an art as well as a science. Bomford,[8] in his Bradshaw Lecture in 1952, discussed the changing concepts of health and disease, and drew attention to the work of Grace, Wolf and Wolf,[9] and to their contributions to the study of man rather than mechanism. These workers showed that while in animal experiment the action of a drug was constant and predictable, there were instances where the effect of the same drug in man depended on the meaning of its administration to a particular person and not on its accepted pharmacological action. In other words, the reaction depended on the emotional relationship of physician and patient. This also confirmed objectively what was common knowledge to psychiatrists, that changes in the colon were greatest in those who appeared to suppress anger. This objective study of changes in the function of the organs of man in health and disease in relation to emotional states and life situations, Bromford regarded as the beginning of a new chapter in the understanding of illness.

The inadequacy of the present theories of psychological causation seems to justify clinical eclecticism, for as McInnes[10] has said, the physician and the patient may differ so much in their natural modes of thought that in attempting to give insight and reveal pathological attitudes, the approach may have to digress from rational intellectual explanations to the use of artistic media by analogy and participation. The misguided advice so often given to neurotic persons to 'pull themselves together' suggests that well-adjusted persons possess an integrating mental capacity, which functions at the level of values and abstract thought. It is interesting to recall that on active service, during the last World War, the ships' companies of submarines and members of air crews were able to endure months of arduous and hazardous duties and were able to integrate these stresses into their general experience without breaking down. This state of high morale was found to depend not only on constitutional factors, but on adequate aims and purpose, good leadership, a sense of discipline, news from home, physical amenities, and the feeling in the individual that he mattered in a group of similar people; yet morale is a psychic phenomenon which belongs to the level of values and is an example of the whole being greater than the sum of the parts.

It therefore seems that any attempt to understand human behaviour by the analytic methods of science in terms of physiology and psychology is explaining the whole in terms of the part. This approach needs to be balanced by increased insight into those aspects of mind which integrate relationships and carry meaning—the need for a personal philosophy of life.

The present concept of psychosomatic medicine indicates a transitional change in medical thought, but the word may itself suggest a false dichotomy and already the phrase 'integral medicine' has appeared in the literature. In the past medicine has enjoyed a unique status because it has considered the whole man. To this end psychiatry may have a contribution to make, although it is not its function to provide a philosophy of medicine. The need for a philosophy of medicine has been ably expressed by Russell Brain,[11] and most psychiatrists would agree with his holistic view that 'man is more than can be explained as a summation of his nervous impulses or the resultant of his conflicting complexes'.

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South African Medical Journal

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EDITORIAL

DIGITALIS TOXICITY

The cardiac glycosides are widely used drugs with great potentialities of producing toxic effects, but serious results from overdosage are relatively infrequent judging by reports in the medical literature and from the experience of physicians generally. The increased use of chemically pure glycosides in the place of the traditional preparations such as the standardized leaf products has emphasized the problem of toxicity. The frequent concurrent use of diuretics is an important factor in inducing digitalis toxicity. The use of adrenocortical steroids, when regarded as necessary in a particular patient with congestive cardiac failure who is receiving digitalis, might conceivably induce alteration in the responsiveness of the cardiac muscle to the glycosides. Potassium depletion produced in these two instances would enhance the toxic effects of digitalis. Calcium in excess also increases the toxic effects of digitalis.

The mode of action of digitalis glycosides on the myocardium is not clearly understood. Many factors may be involved. Amongst others it has been known for many years that the systolic effect occurs only in the presence of calcium ions. Calcium ions and digitalis have a synergistic action on the heart. This has been demonstrated not only experimentally, but clinically also deaths have occurred in digitalized patients who received calcium salts intravenously. Digitalis may act by rendering the heart more sensitive to calcium or by mobilizing calcium ions for the activation of the enzyme adenosinetriphosphatase in heart muscle. The concentration of potassium ions is also affected by the glycosides, toxic doses producing a decrease in the potassium content of heart muscle.

The administration of potassium salts experimentally and clinically has been shown to prevent or abolish digitalis-induced arrhythmias. The use of potassium is not without danger and the greatest care is required because of the depressant action of potassium ions on the heart.

Deficiency of calcium produced in animals has been shown to reduce the toxic effects of digitalis. A hypocalcaemic state can be rapidly induced by intravenous injection of the chelating agent ethylenediamine tetra-acetic acid (EDTA). This has been demonstrated to be of some value in the severely digitalized animal, in which irregular auricular and ventricular contractions and fluctuating blood pressure were temporarily restored to normal.[1] By this controlling action time may be gained for the body mechanisms to clear the glycosides and thus save the life of a severely digitalized

VAN DIE REDAKSIE

DIGITALIS-VERGIFTIGING

Die hart-glikosiede is alombekende middels wat baie maklik vergiftiging kan veroorsaak, maar ernstige gevolge weens oormatige toediening is betreklik seldsaam, as 'n mens moet oordeel na verslae in die mediese literatuur en die ondervinding van geneeshere in die algemeen. Die toenemende gebruik van chemies suiwer glikosiede in plaas van die tradisionele preparate soos die standaard bereide blaarprodukte, het die probleem van vergiftiging beklemtoon. Die feit dat diuretika dikwels tegelykertyd gebruik word, is 'n belangrike faktor by die ontstaan van digitalis-vergiftiging. Dit is moontlik dat die gebruik van bynierskorssteroïede, wanneer dit nodig geag word by 'n besondere pasïent met stuwende hartversaking wat met digitalis behandel word, 'n verandering in die reaksie van die hartspier op die glikosiede teweeg kan bring. Die kaliumvermindering wat by hierdie twee gevalle veroorsaak word, sal die vergiftigende effekte van digitalis versterk. Ook sal 'n oormaat kalsium die vergiftigende uitwerking van digitalis aanhits.

Daar is nog duisterheid oor die wyse waarop digitalis op die hartspier inwerk. Baie faktore is moontlik daarby betrokke. Onder andere is dit reeds jare lank bekend dat die sistoliese effek slegs voorkom as kalsium-ione aanwesig is. Die kalsium-ione en digitalis het 'n sinergistiese aksie op die hart. Hierdie feit is reeds proefondervindelik bevestig, en ook in die kliniek het daar sterfgevalle voorgekom onder pasiënte wat met digitalis behandel was en wat kalsiumsoute binneaars ontvang het. Moontlik werk digitalis deur die hart gevoeliger te maak vir kalsium, of deur die kalsium-ione te mobiliseer vir die aktiveer van die ensiem adenosientrifosfatase in die hartspier. Die konsentrasie van kalium-ione word ook geaffekteer deur die glikosiede, aangesien vergiftigende dosisse 'n vermindering van die kaliumgehalte van die hartspier veroorsaak.

Dit is proefondervindelik sowel as klinies bewys dat die toediening van kaliumsoute aritmie, veroorsaak deur digitalis, kan voorkom of oorwin. Daar skuil egter gevaar in die gebruik van kalium, en dit moet baie versigtig toegedien word, omdat kalium-ione 'n neerdrukkende of stremmende uitwerking op die hart uitoefen.

Dit is reeds bewys dat 'n kalsiumtekort bewerkstellig by diere die vergiftigende aksie van digitalis verminder. So 'n toestand van bloedkalsiumtekort kan vinnig bewerkstellig word deur aarinspuiting van die chelaatmiddel etieleendiamien-tetra-asynsuur (EDTA). Hierdie prosedure het waardevol geblyk by diere wat sterk onder die invloed van digitalis was; die onreëlmatige voorkamer- en kamersametrekkings en wisselende bloeddruk is op hierdie wyse weer tydelik op normaal gebring.[1] Deur so 'n beheeraksie kan die liggaamsmeganismes kans kry om die glikosiede weg te ruim, en die lewe van 'n digitalis-vergiftigde pasiënt kan so

individual. Nice adjustment of the dose is necessary. Too big a dose of the chelating agent injected rapidly sometimes produces depression of the myocardium with a marked fall of blood pressure. The action of the chelating agent may be explained on the basis of its combination with calcium and alteration of the calcium-potassium ratio.

In a clinical study of digitalis toxicity prompt termination of arrhythmias was induced in 3 patients by the intravenous administration of the disodium salt of EDTA, and in 2 others the previous administration of this compound in one patient and of the magnesium salt in the other significantly increased the dose of digitalis glycosides required to produce major arrhythmias.[2] The circulating serum calcium was not actually lowered but the fraction bound (unionized) to the chelating agent was physiologically not available. Much depends on the dose; large doses can produce hypocalcaemic tetany. The authors administered 600 mg. of the disodium salt of EDTA (in 250 ml. of 5% dextrose in water) intravenously over a period of half an hour, but larger doses may also be given.

Both supraventricular and ventricular arrhythmias induced by digitalis (incidentally also ectopic beats not caused by digitalis) were abolished by the chelating agent. Control of the arrhythmias removed their aggravating effect on the heart failure.

The desirability of an antidote for use in severe intoxication with digitalis is obvious. No satisfactory antagonist has hitherto been available. Potassium salts are not without danger. The use of EDTA would appear to offer the advantages of prompt action and greater safety.

1. Sapeika, N. (1954): Arch. Int. Pharmacodyn., **97**, 373.
2. Gubner, R. S. and Kallmann, H. (1957): Amer. J. Med. Sci., **234**, 136.

gered word. Die dosis moet egter baie sekuur bereken word. As te veel van die chelaatmiddel vinnig ingespuit word, veroorsaak dit soms stremming van die miokardium en 'n skerp daling in die bloeddruk. Die aksie van die chelaatmiddel kan verduidelik word op die basis van sy verbinding met kalsium en verandering van die kalium-kalsium verhouding.

In 'n kliniese studie van digitalis-vergiftiging was die aritmie by 3 pasiënte dadelik beheer deur binneaarse toediening van die dinatrium-sout van EDTA; by 2 ander het die voorafgaande toediening van hierdie verbinding by die een, en van magnesiumsout by die ander, die dosis van digitalis-glikosiede nodig om ernstige aritmie te veroorsaak aansienlik opgeskuif.[2] Die sirkulerende serum-kalsium was nie werklik verminder nie, maar die fraksie onverbind (ongeïoniseer) met die chelaatmiddel was fisiologies nie te vinde nie. Baie hang van die dosis af—groot dosisse kan tetanie weens hipokalsemie veroorsaak. Die skrywers het 600 mg. van die dinatrium-sout van EDTA (in 250 ml. van 5% dekstrose in water) binneaars toegedien oor 'n tydperk van 'n halfuur, maar groter dosisse is ook toelaatbaar. Die chelaatmiddel het beide die supraventrikulêre en die ventrikulêre aritmie wat deur die digitalis veroorsaak was, beheer (en terloops ook ektopiese kloppings wat nie hieraan te wyte was nie). Die beheer van die aritmie het dan ook verhinder dat die onreëlmatighede die hartstoornis vererger.

Dit is duidelik dat 'n teenmiddel vir gebruik by ernstige digitalis-vergiftiging dringend nodig is. Dusver was daar nog geen bevredigende weermiddel nie. Die kaliumsoute is nie heeltemal veilig nie. Skynbaar bied die gebruik van EDTA die voordele van vinnige uitwerking en groter veiligheid aan.

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NAUDÉ VERSUS WHITTLE

This action came to trial at Grahamstown in the Supreme Court (Eastern Districts Local Division) in February 1957 and judgment was delivered on 29 April 1957. The judgment was published in this *Journal* on 8 July 1957.

The plaintiff is Dr. W. J. Naudé who is in practice at Franklin and is District Surgeon for the Franklin and Zwartberg areas of the Mount Currie District, and the defendant is a farmer in the Zwartberg area who is a member of the Zwartberg Farmers' Association. Dr. Naudé sued the defendant for damages in respect of the publication by the defendant of a certain statement made at the annual meeting of the Zwartberg Farmers' Association held on 27 October 1955, when over 50 persons were present. The statement was to the effect that when a suicide was committed 'on the night of 13 October 1955 the District Surgeon, Franklin (meaning and referring to the plaintiff), was telephoned at 10.30 p.m. and refused to appear at that hour although the circumstances were explained'. The statement was in fact untrue, for Dr. Naudé was never notified of the suicide during the night on which it occurred or received a request that he should go out to see the body. Dr. Naudé suffered both socially and professionally, and his practice deteriorated sharply after the meeting at which the statement was made.

The learned Judge said: 'It is clear that the statement about plaintiff . . . was a highly misleading one which conveyed implications altogether untrue, implications which defendant Naudé has said he intended to convey. These implications were of a defamatory nature and there has been no attempt on the part of the defendant to show that they were not defamatory. The only serious defence to the resultant claim for damages is that of qualified privilege . . . ' In dismissing the case with costs the learned Judge said that he considered the defendant's statement was made on a privileged occasion without malice and in the *bona fide* belief that it was true; and added: 'If, however, I am wrong in finding that the occasion of its publication was privileged, I would after consideration of all the circumstances . . . allow in damages the sum of £800.'

Although Dr. Naudé had morally succeeded in his action, the decision had gone against him on a question of law. He had cleared his good name and reputation but, apart from the damage he had suffered from the defamatory statement, he must have sustained a heavy financial loss from the action.

At that stage the matter was one for grave concern to the medical profession, for in the light of the judgment it appeared that medical practitioners (as well as other members of the

community) would find that the circumstances under which defamatory statements might be made against them with impunity were much wider in scope than they had hitherto supposed.

The judgment, however, has been reversed on appeal. Dr. Naudé's appeal was heard by the Appelate Division on 3 December 1957 and the judgment of Chief Justice Fagan delivered on 9 December is published in the present issue of the *Journal* (p. 80). The fact was not disputed that Dr. Naudé had not been informed of the suicide and had not been asked to go and see the body, and the case turned on the issue of privilege. The Chief Justice towards the end of his judgment said:

'The impression I get is that the respondent worked himself up into such a state of annoyance when he . . . failed to get the immediate response he apparently expected from the telephone service, the police and the District Surgeon that he did not even try to form a calm and dispassionate judgment but decided to have them all taught a lesson. It seems to me to have been that frame of mind, and not a sense of duty, which led him to put on the message he had received a sinister construction not warranted by its actual content, and then to pass his construction on to the farmers' meeting as if it was the actual wording of the message. That was an abuse of whatever privilege the occasion might have afforded him.

'It follows that the appeal should succeed . . . .'

The appeal was allowed and damages awarded to Dr. Naudé in the sum of £800 with costs.

Dr. Naudé is to be congratulated on the result of his action, which is to be regarded as having been fought in vindication of the medical profession as a whole, and not only of himself. It is gratifying to note the support which was afforded to Dr. Naudé by the Transkei Branch of the Medical Association and its East Griqualand and East Pondoland Divisions.

DIAGNOSIS AT THE SHOULDER*

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Symptoms arising from the tissues at the shoulder are seldom felt at the shoulder itself. The exception is the acromio-clavicular joint. This is developed within the fourth cervical segment and cannot therefore refer pain to the arm. All the other common lesions at the shoulder affect structures derived largely from the fifth cervical segment. Hence the pain is often felt to start at the lower deltoid area and to spread to the end of the relevant dermatome, i.e. to the radial side of the wrist. Rarely it radiates as far as the base of the neck as well.

How far the pain is referred depends on the severity of the lesion. For example, in slight arthritis or tendinitis, the pain is usually felt at the upper arm only, whereas the same lesion, should it become more intense, leads to radiation of pain as far as the wrist. This extended reference is an error of perception occurring in the sensory cortex. A minor impulse stimulates a few cells in the appropriate part of the cerebral mosaic of cells corresponding to the relevant dermatome; a major impulse affects a larger number of cells, even perhaps every cell belonging to that dermatome. If so, the pain is felt over a wide area, but does not spread to the adjacent mosaic that corresponds to the segments on either side. Precise delineation of the extent of the pain thus has two values: (*a*) It outlines the dermatome and thus shows the examiner within which segment to look for the lesion; (*b*) it indicates the severity of the pathological process.

Whatever the disorder at the shoulder may be, the pain is apt to be felt at the same place. For example, whether arthritis, bursitis or tendinitis is present, the lesion lies in a structure of largely fifth cervical derivation; hence fifth cervical reference is common to each. Apart, therefore, from the pain at the very point of the shoulder that suggests the acromio-clavicular joint, where in the arm the pain is felt has no significance, and its extent of distal reference indicates severity rather than provenance.

Possible Lesions

Pain in the shoulder and arm has 8 possible sources:

1. The shoulder joint.
2. The subdeltoid and subcoracoid bursae.
3. The muscles and tendons about the shoulder.
4. The acromio-clavicular joint.
5. The costo-coracoid fascia.
6. The humerus and scapula.
7. By reference distally from the tissues forming the base of the neck and upper thorax, including the cervical nerve-roots, the subclavian artery, the lower trunk of the brachial plexus, the first rib, the heart and the diaphragm.
8. By reference proximally from the nerve-sheaths in the arm, at the elbow or at the wrist.

EXAMINATION

The first question is: Does the pain felt at the shoulder arise from the tissues about the shoulder? The second is: If the shoulder is at fault, which of the structures there contains the lesion? The first part of the examination decides the former point, and since any patient with arm ache is complaining of pain felt within a dermatome between the fourth cervical and the second thoracic, these segments must be examined from beginning to end. The first stage of the examination, therefore, consists of a quick survey from neck to hand. If a lesion is found present in the shoulder area, this is examined more carefully next. If the pain is found to be referred to the shoulder from some other moving part, examination is concentrated there. If no abnormality, i.e. neither limited movement, pain nor weakness, is found at all, the lesion clearly lies outside the moving parts, and conditions like angina or diaphragmatic pleurisy are brought

* A paper presented at the South African Medical Congress, Durban, September 1957.

to mind. If all the movements are stated to hurt, or a number of contradictory movements, the question arises of a psychogenic disorder.

The examination proceeds as follows, the patient being asked as he performs each movement if it hurts and, if so, where, while the examiner notes if weakness is apparent on any of the resisted movements:

Neck:	Active flexion, extension Both side-flexions Both rotations
Scapula:	Active elevation (C3)
Shoulder:	Active elevation (C5)
Elbow:	Passive flexion and extension Resisted flexion (C5 and C6) and extension (C7)
Wrist:	Resisted flexion (C7) and extension (C6)
Thumb:	Resisted extension (C8)
Finger:	Resisted adduction of fourth and fifth fingers (T1)

If this examination shows that pain or weakness was elicited only on movement of the shoulder, this is examined in detail. However, if abnormality is detected on movements other than those at the shoulder, naturally the lesion is shown to lie elsewhere.

The Twelve Movements

During the clinical examination of the shoulder, 12 tests must be made. The examiner merely muddles himself if more than 12 are carried out; if less than the 12 are tested his examination is incomplete. These 12 are:

1. *Active Elevation.* The patient is asked to bring his arm up as high as he can and is asked what he feels. His active range of movement and statement on pain are noted for correlation later.

Patients do not know how the arm gets up to full elevation; most imagine that 180° of movement are present at the shoulder joint. In fact the first 90° of elevation take place at the scapulo-humeral joint. The next 60° result from rotation of the scapula. The last 30° involve adduction of the humerus, the surgical neck crossing in front of the coracoid and acromion processes, which rotation of the scapula has now made to point upwards instead of forwards. This knowledge is important in detecting psychogenic limitation of active elevation. If the scapula is mobile and its muscles intact, even if the shoulder is ankylosed, 60° of active abduction must be attainable by scapular movement alone. Since neurosis is often accompanied by limitation of abduction of the arm, any inconsistency between the findings on active, passive and resisted abduction must not be overlooked.

2. *Passive Elevation.* The examiner pushes the patient's arm up as high as possible and notes (*a*) whether full elevation is obtainable; (*b*) if it hurts; (*c*) if active and passive elevation correspond in range or not. With some experience he also notes that in patients with a normal joint the end of elevation has a characteristic free feel; whereas in capsular contracture the movement comes to a dull stop, rather as if a piece of tough leather were being unsuccessfully stretched.

3. *Scapulo-humeral Range.* The examiner fixes the lower angle of the scapula with his thumb applied to the patient's mid-thorax and lifts the elbow outwards with his other hand until he feels the scapula start to move. He notes the amplitude of this angle (90° to 100°).

4. *Painful Arc.* This applies only when 90° of abduction range at the shoulder joint is present, passively or actively. The patient is asked to bring his arm up outwards and to state if, at any point in the upward movement, he feels an ache and, if so, if it disappears again or continues unchanged all the way up to full elevation.

5. *Passive Lateral Rotation.* The patient bends his elbow to a right angle and the examiner holds his forearm pointing straight forwards. The humerus is now rotated outwards, first on the good, then on the affected side. The range is usually 90°, occasionally a little more in the young, and often 10° to 20° less in the elderly. If the restriction is due merely to age, it is painless and bilateral.

If the lateral rotation is limited, the angle by which this falls short when the two sides are compared is estimated, and the examiner tests for the capsular feel. The appearance or not of pain when the extreme is reached is noted. Rarely a painful arc on rotation is noted; if so, a tender structure is being pinched.

6. *Passive Medial Rotation.* The normal range, starting from the forward position of the forearm (as above) is 90°. The examiner rotates the patient's humerus inwards and notes if full painless, full painful, or limited range is present, and in the latter case assesses the amplitude of this limitation (0° to 100°).

7. *Resisted Adduction.* The patient's elbow is brought a few inches from his side and he is then asked to pull his arm to his side as hard as he can. The examiner prevents all movement by placing one hand on the inner side of the patient's elbow, and the other on the patient's flank. The patient states if pain is evoked or not, and if so, where. The examiner notes the strength of the muscles.

8. *Resisted Abduction.* The patient pushes his elbow laterally as hard as he can; the examiner prevents all movement by one hand applied to the outer side of the patient's elbow, steadying himself with his hand at the far side of the patient's trunk. The patient states whether he feels pain or not; the examiner notes power.

9. *Resisted Lateral Rotation.* The patient bends his elbow to a right angle, the forearm pointing forwards. Keeping his elbow well into his side actively, he tries to rotate the arm outwards against the examiner's pressure applied to the patient's lower forearm (not his hand), so strongly that the shoulder joint does not move. Pain is reported; strength noted.

10. *Resisted Medial Rotation.* The same as for lateral rotation, except that the resisted movement takes place towards the trunk.

11. *Resisted Extension of the Elbow.* The patient's elbow is bent to a right angle; he then presses his forearm down against the examiner's resistance applied to his lower forearm (not the hand). Pain is mentioned; strength assessed.

12. *Resisted Flexion of the Elbow.* The same as for resisted extension, except that the patient flexes his elbow.

Radiography

Clinical examination is now complete. Those who wish it, may now inspect the radiograph, but must not expect very much help from it, and must be careful not to be misled.

Fracture, tuberculosis, neoplasm, neuropathic arthropathy, chondromatosis, and calcification are revealed. But in the ordinary case of arthritis, bursitis or tendinitis, the photograph reveals no abnormality. Moreover, the presence of a small osteophyte or an area of calcification does not imply that either is the cause of a patient's symptoms. Hence radiographic signs should be given weight only if they correspond with the findings on clinical examination. If the clinical signs suggest severe trouble, X-ray examination is of course essential, and shows the lesion clearly. If a calcified deposit is seen within the structure already singled out as that at fault considerable significance attaches; for its presence alters the treatment.

INTERPRETATION

When the pattern that emerges on clinical examination comes to be interpreted, there are 4 main divisions:

1. Limited passive movement: capsular pattern, i.e. arthritis.
2. Limited passive movement: non-capsular pattern.
3. Full passive range: one resisted movement hurts, i.e. a tendinous lesion.
4. Full passive range: weakness of one or more muscles.

CAPSULAR PATTERN: ARTHRITIS

The capsular pattern at the shoulder is: So much limitation of abduction, more limitation of lateral rotation, less limitation of medial rotation. Probable proportions are:

Arthritis	*Abduction limited by*	*Lateral rotation limited by*	*Medial rotation limited by*
Slight ..	10°	30°	full and painful
Medium ..	45°	60–70°	15–30°
Gross ..	70–80°	90°	45°

If clinical examination discloses the capsular pattern, arthritis is present. Five types exist:

1. *Traumatic Arthritis*

A minor injury is reported by a middle-aged or elderly patient. After the immediate pain has ceased, he feels little or nothing for some days, then the upper arm begins to hurt, first on certain movements only; later a constant ache sets in, soon spreading down to the elbow. Untreated, the pain and limitation of movement increase in the same way as for a freezing arthritis, but can be prevented by passive movement, started as soon as possible after the accident and continued daily until the full range has been restored. By contrast, freezing arthritis cannot be aborted however early treatment is started.

2. *Freezing Arthritis*

This is a remarkable phenomenon, without parallel at any other joint. Freezing arthritis usually comes on between the ages of 45 and 55. It follows, with only slight variations between one individual and another, a fixed course. 'Freezing arthritis' is a good descriptive label; for after freezing, the joint then thaws.

For no apparent reason, a middle-aged patient begins to feel an ache at the shoulder on moving the arm. There is no pain when the arm is kept still. At this time examination reveals almost a full range of movement at the shoulder joint, each extreme hurting when tested passively; the resisted movements prove painless. After a month or two, the pain on movement becomes more severe and spreads as far as the elbow; a constant ache in the arm appears, worse at night and worse still if the patient lies on that side in bed. Limitation of movement at the shoulder joint of the capsular type is now clearly apparent. At the end of two or three months the pain has become constant and severe, reaching to the wrist. Severe pain on the slightest jarring of the joint may compel the patient to wear a sling. Examination now shows an abduction range of only 30° to 45° with corresponding limitation of rotation. The shoulder, however, never becomes strictly speaking 'frozen'; for complete fixation of the scapulohumeral joint (which may occur in infective arthritis) is absent. No diminution in the range of movement takes place after four months.

At the end of 4 months the pain begins to ease and at the end of 6 months the constant ache has largely ceased. Once more pain is felt only when the shoulder is moved and it leaves the forearm, remaining only in the arm. The patient begins to be able to lie on that side again at night. Later still, the pain produced by movement becomes confined to the deltoid area and the range of movement begins to return to the joint. At the end of 10-16 months the patient is well; the ache has ceased and he has regained a full range of movement at the shoulder.

3. *Infective Arthritis*

This is the name given to rheumatoid arthritis when it affects one large joint only. The sedimentation rate is usually normal. It must be distinguished from a freezing arthritis because it responds excellently to hydrocortisone; whereas this is ineffectual in freezing arthritis. If trophic changes in the hand complicate an arthritic shoulder, the condition goes by the name of 'shoulder-hand syndrome'.

The distinguishing points are as follows, but in cases of doubt it is best to inject hydrocortisone into the joint and observe the result.

(*a*) *Age.* In freezing arthritis the patient is usually aged between 45 and 55, with extremes of 40 and 60. Infective arthritis can occur at any age, but in fact I have not yet met with a patient with monarticular involvement at the shoulder aged less than 25.

(*b*) *Other Joints.* If there is a past history of rheumatoid arthritis in other joints, the suggestion is obvious. Freezing arthritis has never, in my experience, affected the same shoulder twice, but I have known it appear at the other shoulder later; it is very rarely on both sides at the same time. Spondulitis ankylopoietica may be heralded by infective arthritis at the shoulder, or may be complicated by spread there, after the sacro-iliac joints have already become affected.

(*c*) *Too much Limitation.* Freezing arthritis usually stops getting worse when abduction has become 45° limited. Rarely 60° limitation of abduction is reached. Hence, less than 30° abduction range suggests infective arthritis.

(*d*) *Wrong timing.* If there is a marked discrepancy between the time that has elapsed since the onset and the known chronology and course of freezing arthritis, the lesion is probably infective.

(*e*) *Muscle Wasting*. If the deltoid is wasted, a joint lesion more severe than a mere freezing arthritis is present.

4. *Osteo-arthritis*

This diagnosis cannot be made by radiography. Osteo-arthritis may be absent clinically in a patient who has an osteophyte at the humeral head—the pain being due to some other lesion—or it may be present even when the photograph reveals nothing.

As a rule, osteo-arthritis in an elderly patient's shoulder is really a traumatic arthritis. The osteo-arthritic condition of the joint has rendered it sensitive to any slight injury, which lights up the arthritis. Occasionally, painful capsular contracture accompanied by crepitus appears spontaneously. If one shoulder crepitates, the other nearly always does too; this finding is consistent with a full range of painless movement at the osteo-arthritic joint.

5. *Specific Arthritis*

In tuberculosis the joint is completely fixed and the radiograph shows gross changes; in septic arthritis, fever is present; in neuropathic arthropathy there is a painless bony block with huge osteophyte outcrops that show clearly on the radiograph.

NON-CAPSULAR PATTERN

The presence of limitation of passive movement in other than the capsular proportions shows that a lesion other than arthritis is present. This cannot be a tendinous lesion, since it is anatomically impossible for a tendinous lesion of itself to limit passive range, though pain on voluntary movement may deceptively restrict the active range; those who test the range of passive, as well as active, movement cannot be deceived by this reluctance. The point is worth making, since the published views, shared apparently by all orthopaedic surgeons, are to the contrary. A wider adoption of diagnosis by the correlation of the responses to active, passive and resisted movement would help to correct this fundamental error.

Ten possible causes for non-capsular pattern exist; these are: Acute subdeltoid bursitis; Local neoplasm; Pulmonary neoplasm; Capsular adhesion; Subcoracoid bursitis; Contracture of the costo-coracoid fascia; Acromio-clavicular joint-strain; Fracture of first rib; Clay-shoveller's fracture; Hysteria; as follows:

1. *Subdeltoid Bursitis*

This is the common cause of marked limitation of movement coming on in the course of a few days. It is a relapsing condition, often bilateral, usually appearing at one or other shoulder, every 2-5 years.

The radiograph may or may not show calcification; moreover, this is often seen at the other shoulder too, even if no symptoms have as yet arisen there. Between attacks the calcification remains; after 2 to 10 years it may disappear spontaneously, but without rendering the shoulder immune to further attacks of bursitis. Symptomless calcification in this bursa may exist in a patient suffering from some unconnected lesion at the shoulder.

During the acute stage, there is severe constant pain from shoulder to wrist; as the bursitis dies down the pain becomes shorter and finally pain is felt only near the shoulder on movement. The attacks are self-limiting; there is a good deal of pain for a week, then this eases, spontaneous cure taking from 4 to 6 weeks.

(*a*) *The Pattern*. Marked limitation of scapulo-humeral abduction range exists, associated with a full range of passive rotation. Rotation may be painful at its extreme, but the full range can be achieved. This rules out arthritis and brings acute subdeltoid bursitis to mind at once, since in arthritis lateral rotation is more limited than abduction. Other signs are:

(*b*) *No Muscle Spasm*. There is no joint lesion; hence no involuntary muscle spasm appears, limiting the range tested. The patient merely stops the movement actively when it begins to hurt too much and, if asked to bear the pain, can let the arm go a good deal further.

(*c*) *No Muscle Wasting*. Severe arthritis gives rise to wasting, but bursitis does not.

(*d*) *Tenderness*. The bursa is very tender all over its palpable extent.

(*e*) *Painful Arc*. During the attack, not enough abduction range exists to enable a painful arc to appear. When recovery is well advanced, and the range is restored, a painful arc can be elicited during the last week or so.

Chronic subdeltoid bursitis does not result from an acute bursitis that has not cleared up completely; it is a separate disorder.

2. *Local Neoplasm*

The neoplasm is nearly always secondary and infiltrates bone and muscle equally. Hence marked limitation of movement co-exists with obvious muscular weakness. If, as is usually the case, the patient appears with a recent normal radiograph, this should be repeated, say, a fortnight later.

3. *Pulmonary Neoplasm*

Any serious lesion is apt to produce spasm in near-by muscles. For example, a Brodie's abscess at the upper tibia sets up muscle spasm limiting movement at the knee joint; appendicitis causes rigidity of the abdominal muscles and so on.

When a pulmonary neoplasm lying antero-laterally reaches the ribs, the pectoralis major muscle tends to go into spasm. Hence the scapula is found fully mobile actively; there is a full range of scapulo-humeral abduction actively, but the patient cannot elevate his arm beyond the horizontal, nor can the examiner passively. When the reason for this discrepancy is sought, elevation of the arm beyond the horizontal is found restricted by involuntary contraction of the pectoralis major muscle. The patient is apt to arrive bringing with him a radiograph revealing no abnormality at the shoulder; the lungs should be X-rayed.

The same signs are also found in contracture of the pectoral scar after radical mastectomy.

4. *Capsular Adhesion*

This is uncommon; for traumatic arthritis is the usual sequel to a joint injury. The cause is usually a dislocation, which has severely torn the anterior aspect of the joint capsule. After reduction, this tear heals, but by some months later a large scar has formed, which contracts slowly. In the end this is apt to restrict lateral rotation only; hence the pattern is: 10° to 20° painful limitation of lateral rotation; full elevation; full medial rotation. Only two other conditions have this

pattern: subcoracoid bursitis (see below) and a ruptured infraspinatus tendon. In the latter case, the localized capsular contracture is a disuse phenomenon coming on after a year or so. When the resisted movements are examined, lateral rotation is found powerless.

5. *Subcoracoid Bursitis*

This is also rare, and confusingly enough gives rise to isolated limitation of lateral rotation, as does anterior capsular contracture (see above) and rupture of the infraspinatus muscle. In the latter event, power is found lost on resisted lateral rotation.

Differential diagnosis rests on (1) the absence of a history of a severe injury to the front of the joint; (2) the absence of the capsular feel and of spasm limiting the amount of lateral rotation range (the patient can, by disregarding the pain, allow rather more movement); (3) if the humerus is abducted to the horizontal, a full range of lateral rotation can be achieved in bursitis, but not, of course, without pain. In capsular contracture, it is unattainable whatever the position of the arm.

6. *Contracture of the Costo-coracoid Fascia*

This is a very common cause of limited elevation of the arm. The symptom is gradually increasing upper pectoro-scapular pain on one side only. It is provoked at first only by full elevation of the arm. After a year or two elevation becomes slightly limited, and any prolonged reaching upwards leads to some hours' or days' increased aching.

The syndrome is difficult to recognize; for the symptoms suggest a cervical-disc lesion and the signs unless carefully studied suggest a psychogenic disorder. The key to the condition is the discovery of slight limitation of elevation of the scapula.

Examination of the neck movements reveals that active side-flexion away from, and resisted side-flexion towards, the painful side hurt at the root of the neck. Upward movement of the scapula, active or passive, is painful at its extreme and slightly limited. Resisted elevation is painless. Forward movement of the scapula is usually full and painful; backward full and painless. Active and passive elevation of the arm is about 10° limited by pectoro-scapular pain, but the passive range of movement at the gleno-humeral joint is neither restricted nor painful. All the resisted shoulder movements hurt a little at the base of the neck.

This curious pattern occurs only in contracture of the costo-coracoid fascia. Inspection of the range of elevation of both scapulae together discloses limitation on the painful side and this rare condition is brought to mind. One cause is dense adhesions at the apex of one lung such as occurs in long-standing tuberculosis. However, not all cases show such a shadow. Follow-up for several years has not revealed the cause, and it would seem that in middle age unilateral idiopathic contracture can come on. The condition appears to become stationary after some years, and gross restriction of elevation has not been encountered.

7. *Acromio-clavicular Joint Strain*

In theory, the scapular movements should all hurt at their extremes, but sometimes they do not and the pattern that then emerges is rather puzzling. A full range of movement is found at the shoulder joint, each extreme hurting at the point of the shoulder. The resisted movements prove painless. Each extreme of passive movement hurting suggests arthritis; a full range of movement negatives arthritis. This contradiction calls to mind the acromio-clavicular joint. If its superficial aspect is affected, tenderness is found there and no difficulty arises. If, however, the deep aspect of the joint is affected alone, the upper surface of the joint is not tender and there is a painful arc; hence a typical subdeltoid bursitis is closely mimicked. In some cases differential diagnosis is all but impossible clinically, and can be established only by local anaesthesia.

Ankylosis of the acromio-clavicular joint is rare and occurs in spondylitis deformans. The arm can then be abducted to the horizontal only, passively or actively, but all the movements at the scapulo-humeral joint are of full range and painless. The cause of this discrepancy becomes obvious when the examiner notes that the scapula cannot move appreciably on the chest wall.

8. *Fracture of the First Rib*

This may be a stress fracture, without history of trauma. The pain is at one side of the base of the neck and is brought on by the neck and scapular movements. Voluntary elevation of the arm stops at the horizontal, but a full passive range exists at the shoulder. The radiograph is diagnostic.

9. *Clay-shoveller's Fracture*

This is usually a traction fracture. The pain is at the centre of the lower neck. Though the neck movements scarcely hurt, the patient can hardly abduct either arm actively at all. The passive range is full. The radiograph shows avulsion of the tip of the spinous process of the seventh cervical or first thoracic vertebra.

10. *Hysteria*

The shoulder joint is closely connected with the emotions, pleasure being exemplified by the outstretched hand and repugnance by drawing the arm in to the side. Limited active abduction at the shoulder is therefore not uncommonly a withdrawal phenomenon in conversion hysteria. But the patient does not realise that, unless the scapula is also fixed, mobility of this bone permits considerable abduction. Even in ankylosis at the shoulder joint, 60° of movement of the arm away from the side are possible, actively and passively, owing to scapular rotation. Hence detection of psychogenesis is simple if the range of voluntary and passive elevation is contrasted with the range of passive abduction at the scapulo-humeral joint. In organic disability, the range of passive elevation equals the passive scapulo-humeral range plus 60°.

The large number of patients who have carried off this psychogenic conversion undetected, and have in consequence enjoyed years of treatment, testifies afresh to the diagnostic importance of comparing the responses to active, passive and resisted movements.

TENDINOUS LESIONS

These are common at the shoulder, but it is by no means enough merely to call them rotator cuff lesions. Which member of the cuff, and what part of it, must be discovered. Two criteria must be satisfied before a diagnosis of a tendinous lesion can be made: (1) There must be a full range of passive movement at the shoulder joint. However, there is nothing inconsistent in pain being evoked by stretch or pinch at one extreme. (2) Pain must be evoked when one group of muscles

is tested against resistance, but there must be no pain when all the other groups are similarly tested. Accessory tests then serve to show which member of the muscle group contains the lesion and, often, which part of it. If all the resisted movements are found painful, the conclusion is drawn that the muscles are unaffected and that the pattern formed by the passive movements should be studied afresh; alternatively, the lesion lies at a joint proximal to that under examination.

1. *Resisted Abduction Hurts*

The patient's arm hangs by his side and the examiner resists the attempted abduction by pressing so hard against his elbow that no movement takes place at the shoulder joint. If this endeavour causes pain (and the other resisted movements do not hurt) the supraspinatus tendon is at fault. In theory, the deltoid muscle might be the source of pain evoked in this way, but in my experience lesions of the deltoid other than a direct temporary bruising do not occur.

The position of the lesion within the supraspinatus tendon is then identified as follows (see Fig. 1).

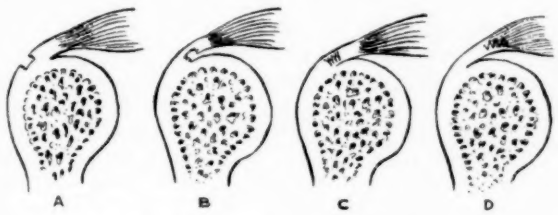

Fig. 1. Supraspinatus tendinitis. Accessory signs indicate the exact position of the lesion.
A. Painful arc. The lesion superficially at the teno-periosteal junction.
B. Pain on full passive elevation. The lesion lies deeply at the teno-periosteal junction.
C. Both a painful arc and pain on full passive elevation. The lesion traverses the distal end of the tendon.
D. Neither an arc nor pain on full passive elevation. The lesion lies at the musculo-tendinous junction.

(*a*) *A Painful Arc Exists.* This shows the lesion to lie where it can be pinched between acromion and greater tuberosity, i.e. superficially near teno-periosteal junction, just medial to the greater tuberosity of the humerus. Should the painful arc prove more marked when the abduction movement is carried out in medial than in lateral rotation of the arm (i.e. palm down or palm up) additional information is afforded. Greater pain when the arm is held in lateral rotation (palm up) suggests that the lesion in the tendon lies anteriorly. Of course, the mere presence of a painful arc exculpates the deltoid muscle.

(*b*) *Full Passive Elevation of the Arm Hurts.* This implies tenderness of the part of the tendon which engages against the glenoid rim; i.e. the deep aspect at the teno-periosteal junction. If this sign is found together with a painful arc, the lesion clearly traverses the distal end of the whole tendon.

(*c*) *The Absence of a Painful Arc* and of pain elicited on full passive elevation naturally suggests a lesion of the supraspinatus at the musculo-tendinous junction, since the belly itself is very rarely affected. Tenderness of the musculo-tendinous junction may be sought, and the two sides compared, deeply within the angle formed by the clavicle and the spine of the scapula. However, local anaesthesia should always be used to verify this diagnosis; for an occasional case of tendinitis at the teno-periosteal junction unexpectedly fails to show either of the two localising signs.

In spite of the intimate relation of the supraspinatus tendon and the subdeltoid bursa, a resisted abduction movement, when properly tested (i.e. without any movement taking place between humerus and acromion), is painless in even acute bursitis.

2. *Resisted Lateral Rotation Hurts*

When a patient is asked to rotate his arm laterally against resistance, he usually abducts his arm at the same time, thus exerting both spinatus muscles. To guard against this, he must be asked to hold his elbow well into his side while pressing his lower forearm outwards against the examiner's resistance. If this endeavour causes pain, the infraspinatus tendon is at fault.

The same accessory tests are then employed as in supraspinatus tendinitis, in order to reveal which part of the tendon is affected. Pain on full passive elevation incriminates the deep aspect and a painful arc the superficial aspect of the teno-periosteal junction.

3. *Resisted Medial Rotation Hurts*

This movement elicits pain from lesions of subscapularis, pectoralis major, latissimus dorsi and teres major muscles. The last-named three muscles are all adductors, whereas the subscapularis muscle is not. It suffices, therefore, to show the absence of pain on a resisted adduction movement to demonstrate that the subscapularis is the muscle affected. If a lesion of this muscle has thus been shown to exist, two further localizing signs should be sought. (1) A painful arc. If this is present the lesion can be confidently ascribed to the uppermost part of the teno-periosteal junction, since only the top of the lesser tuberosity can engage against the coraco-acromial arch. (2) Pain on passive adduction across the front of the chest. At the extreme of this movement the lesser tuberosity is squeezed against the coracoid process. This shows the lesion in the tendon to lie in its humeral insertion at its lower extent.

4. *Resisted Adduction Hurts*

The arm is brought a short distance away from the body and the adduction movement resisted by pressure exerted against the inner side of the elbow. The muscles responsible are the pectoralis major, the latissimus dorsi and the two teres. Pain, except when it arises from the axillary portions of the pectoralis major or latissimus dorsal muscles, is usually correctly appreciated by the patient at the anterior or postero-lateral aspects of the thorax respectively. When the patient's sensations are no guide and an adduction movement hurts, the next part of the examination is to ask him to swing his arm first forwards then backwards against resistance. If the former hurts, the pectoral muscle is at fault and confirmation may be sought by asking him to press his hands together. If the backward movement hurts, the fault lies in one of the other three muscles. The teres muscles may be differentiated by the fact that the major is a medial, the minor

a lateral, rotator of the humerus. The latissimus dorsi and teres major muscles being identical in function, no test distinguishes between them.

Palpation follows when the pectoralis major muscle is affected, the fibres just below the lateral half of the clavicle or those at the lower extent of the outer edge being the common sites. The lesion in the latissimus dorsi muscle is usually found at the upper part of the outer edge.

5. Resisted Flexion and Supination of the Elbow Hurt

If the pain is felt at the shoulder, the long head of biceps is at fault. Unless, as is rare, a painful arc exists, no localizing sign is to be found. Hence the tendon has to be palpated along the whole of its extent in the bicipital groove.

PAINFUL ARC

The presence of a painful arc as the arm passes the horizontal on its way up to full elevation means that a tender structure is squeezed as the greater tuberosity of the humerous passes under the acromion and the coraco-acromial ligament.

A painful arc is therefore present in supraspinatus tendinitis and a particularly marked arc suggests calcification in the tendon, in infraspinatus tendinitis and in subscapular tendinitis, provided that the lesion lies at the pinchable part of the tendon. It also occurs if the inferior aspect of the acromio-clavicular joint is affected and in most cases of subdeltoid bursitis.

Chronic Subdeltoid Bursitis

This comes on insidiously for no apparent reason and often continues indefinitely. As a rule the radiograph reveals nothing.

The pain is felt at the deltoid area only and clinical examination reveals a painful arc and nothing else. Neither the extreme of any passive movement nor any resisted movement hurts. Localized tenderness must be sought, but this is difficult to assess since the bursa is normally rather tender to deep pressure. Moreover, the affected area may lie under the acromion out of fingers' reach. The only way to be sure that the right spot has been found is to induce local anaesthesia with 5 c.c. of 0·5% procaine. Five minutes later the shoulder is examined again to discover whether or not the arc has disappeared. It so happens that local infiltration is also the effective method of treatment; the injection thus serves a double purpose.

Incomprehensible Pattern. A pattern may emerge that appears self-contradictory. A full range of movement is found present, usually with a painful arc, but when the resisted movements are tested they hurt and when tested again do not hurt, in an erratic manner. This shifting pattern of resisted movements occurs in chronic subdeltoid bursitis. Another misleading pattern is provided by pain on both resisted abduction and resisted lateral rotation, again together with full range and a painful arc. Sometimes this combination means that both tendons are affected; at others, subdeltoid bursitis. The only way to find out is the induction of local anaesthesia repeated if necessary till the right spot is found.

MUSCLE WEAKNESS

The resisted movement is found weak, but does not bring on or increase the pain. This implies a nerve lesion or complete rupture of the tendon. The possibilities are:

1. *Weak Deltoid Muscle.* An axillary nerve palsy is the result of over-stretching the nerve. This is apt to follow a dislocation of the head of the humerus, which may be momentary, with the result that the typical history of dislocation and reduction is not reported. The deltoid muscle remains weak and wasted, until spontaneous recovery is established in 6-12 months.

2. *Powerless Supraspinatus Muscle.* This results from rupture of the tendon. The patient cannot abduct his arm voluntarily from his side, since the deltoid muscle merely pulls the head of the humerus upwards into the gap formed by the absent supraspinatus tendon, where it becomes fixed.

Passive abduction meets a marked painful arc at the horizontal. Once this point is passed, the patient finds to his surprise that he can elevate his arm actively to the vertical without difficulty.

3. *Powerless Lateral Rotator Muscles.* The tendons of the infraspinatus and teres minor muscles appear to rupture together, since full lateral rotation becomes impossible, even actively, without resistance. The lesion is rare and the patient is little concerned; except in heavy workers the disablement is minimal.

As a result of never laterally rotating the arm, anterior capular contracture from disuse sets in painlessly in the course of the next year or two, permanently limiting that range.

4. *Weak Supraspinatus and Infraspinatus Muscles.* This is the result of neuritis of the suprascapular nerve. The symptoms are continuous pain in one scapular area, unaltered day or night or by any movement of the neck, scapula or arm. The pain eases spontaneously in 2-3 weeks, but the muscle palsy may take 6 months to recover.

5. *Weak Deltoid, Spinati and Biceps Muscles.* This indicates a fifth cervical root palsy, very probably the result of a fourth cervical disc lesion. The biceps jerk is usually sluggish or absent and the brachioradialis jerk absent or inverted.

6. *Weak Serratus Anterior Muscle.* This is caused by a long thoracic neuritis. The patient may have a fortnight's continuous pain in the scapular area, but at times the palsy appears without any preliminary symptoms. Examination shows that the patient has lost the last 30° to 40° of active elevation, but that there is no bar to full passive elevation. When the muscles about the shoulder are tested, all are found strong; it follows that the defect lies in active rotation of the scapula. When the patient is asked to bring his arm forward to the horizontal and press his hand against the wall, winging of one scapula becomes obvious. Spontaneous recovery in the course of 4-8 months is the rule.

SUMMARY

The shoulder is examined by testing active, passive and resisted movements. The diagnosis is arrived at by correlating these clinical findings and interpreting the pattern that emerges on a basis of applied anatomy.

The illustration is taken from volume I of my *Text-book of Orthopaedic Medicine*, published by Cassell.

FRACTURE OF THE HYOID

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Fracture of the hyoid bone is a rare condition. Stimson,[1] in 1910, collected 45 cases from the literature but most of these resulted from judicial and suicidal hanging and were accompanied by fractures of the thyroid and cricoid cartilages. The remainder occurred before the use of X-rays and were not proved. Poppel and Christman[2] state that in the last 20 years there have only been 7 reports in the literature, to which they add a case of their own. Apart from hanging and strangulation, fracture of the hyoid is thus exceedingly uncommon. The following case was sustained in a motor-car accident.

CASE REPORT

In February 1957, D.P., a Coloured woman aged 55, was a passenger in a car which fell into a ditch. She was sitting in the rear and knocked her chin forcibly against the back of the front seat, and was then dazed. She was brought to Baragwanath Hospital 2 hours later, complaining of pain in the neck. On examination

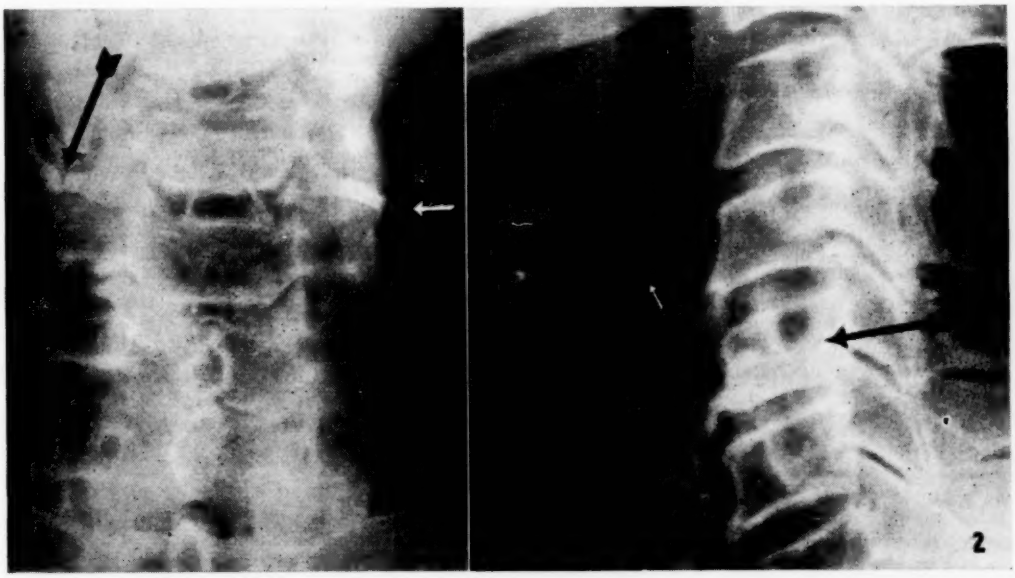

Fig. 1.

Above: Antero-posterior radiograph of the neck. The large arrow points to the fracture through the right greater cornu of the hyoid. The lateral fragment is displaced upwards. The small arrow points to the normal left greater cornu.

Below: Drawing from the radiograph.

Fig. 2.

Above: Lateral radiograph of neck. The large arrow points to the fracture of the body of C4 and the small arrow to the fracture of the right greater cornu of the hyoid.

Below: Drawing from the radiograph.

her neck was stiffly held in a neutral position and could be moved only passively and then but slightly. A small abrasion was present on her chin and she was tender over the anterior and posterior aspects of the upper parts of the cervical spine. X-ray of her neck (Figs. 1 and 2) showed a fracture of the body of the 4th cervical vertebra, and a fracture of the hyoid bone through the right greater cornu.

The patient was given only symptomatic treatment and was not seen again until August 1957. She said that her symptoms cleared up completely after about a month and X-ray now showed normal appearances.

DISCUSSION

There are 3 well-established causes for fracture of the hyoid:

1. Direct injury, as in strangulation, run-over accidents, or a direct blow on the side of the neck.

2. Indirect violence, as in hyperflexion and extension of the neck, as in a fall. Our case would probably fall under this heading.

3. Muscular violence, as in forcible swallowing.

The age of the patient is important because the joints between the body and greater horns of the hyoid disappear by ossification after middle age and this renders the bone more liable to fracture from strangulation.

On X-ray it is important not to mistake the joints between the body and greater horns, for fractures and loss of alignment are the significant finding. The stylo-hyoid ligament ossifies only rarely and is then called the epihyal bone. When the latter is small and is present near the hyoid it may be confused as a fragment from a fractured hyoid. However, normally it is well defined, with a cortical margin.

The commonest symptoms are pain in the neck and throat made worse by talking and swallowing, and dysphagia, especially with solid food.[3]

Complications. Non-union occurs but does no harm.[4] Da Costa[5] states that the fracture should unite in 4 weeks and that change of voice and hoarseness is usually temporary.

Prognosis is good unless a bony fragment perforates the larynx, when there is a danger of oedema glottidis or suffocation from bleeding into the trachea.

Treatment is completely conservative for all cases except where the larynx has been perforated by a bony fragment. The voice should be kept at rest for 1 week and the patient given a liquid diet until he can take solids.

I should like to thank the Superintendent of Baragwanath Hospital for permission to publish and Dr. M. Simchowitz for the clinical notes.

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PRIMARY HYPERPARATHYROIDISM, NEPHROCALCINOSIS AND RENAL STONE

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According to Albright[1] the presence of the parathyroid glands was not known to the world until 1880, when Sandström[2] began a careful examination of the region around the thyroid gland and named the organs which he found, and described as being the size of a small pea, the glandulae parathyroideae. For a good many years the only significance attached to these glands was their anatomical situation.

Von Recklinghausen's Disease

In a Festschrift published in 1891 in honour of the 71st birthday of Rudolf Virchow, Professor von Recklinghausen,[3] of Strassburg, reported on a series of 16 patients suffering from a variety of bone diseases, of which 3 exhibited bone changes which he described as osteitis fibrosa cystica. Subsequent examination by Albright[1] of the data in connection with these 3 cases has led him to conclude that in fact only one was a true case of osteitis fibrosa cystica and that the other 2 were cases of polyostotic fibrous dysplasia. The historic case was a 40-year-old mason, who in April 1888 fell from a 3-meter-high ladder, sustained a fracture, and was admitted to hospital. While he was in hospital a series of spontaneous fractures occurred, and later he developed widespread excruciating pain in the bones and the most extensive bending of several long bones. He died on 4 October 1889, and post-mortem widespread fibrosis, cysts and giant-cell tumours were found in the bones. In 1933 Jung[4] (quoted by Thannhauser[5]) looked up the original autopsy report on this case at the Pathological Institute of Strassburg and found the following comment: 'Above the left thryoid gland, a lymph gland, red-brown in colour, is present.'

In 1903 Askanazy[6] described a case of von Recklinghausen's disease (osteitis fibrosa cystica) and commented upon the presence of a parathyroid tumour. No special significance seems to have been attached to the association. In 1906 Erdheim,[7] working with parathyroidectomized rats, focused attention on an interrelationship between the parathyroids and calcium metabolism. In 1907 the same worker described enlargement of multiple parathyroids in patients dying of osteomalacia[8] and concluded that the enlargement of the parathyroids in osteomalacia is compensatory. Descriptions of parathyroid hypertrophy in osteomalacia continued, and the same line of thought was applied to findings of parathyroid adenomata in cases of osteitis fibrosa cystica; the possibility was not contemplated at that time that what is true for osteomalacia might not be true for von Recklinghausen's disease.

In 1915 Schlagenhaufer[9] pointed out that in von Recklinghausen's disease of bone the parathyroid enlargement involves only one gland, which would be hard to explain if the hypertrophy were secondary. There, however the matter seems to have rested until, in 1925, Felix Mandl,[10] still acting on the assumption that the parathyroids enlarged to meet the greater demand for calcium, transplanted 4 parathyroid glands into the abdominal wall of a man with generalized osteitis fibrosa. No improvement followed, and he accordingly explored the neck, where he found a parathyroid tumour measuring 2·5 × 1·5 × 1·2 cm. This was removed and the subsequent disappearance of pain in the bones, the reduction of excretion of calcium in the urine, and the general improvement in the patient's health, constituted the first clinical evidence that in this disease the parathyroid lesion is primary and the skeletal changes secondary. In 1926 a diagnosis of hyperparathyroidism was made for the second time, when Du Bois *et al.*[11] finding a raised serum calcium in a case of osteitis fibrosa cystica, made a metabolic study which confirmed the suspicion of hyperparathyroidism. In this case after 6 negative neck explorations a parathyroid tumour was eventually found by splitting the sternum.

Renal Stone

Most of the cases of hyperparathyroidism subsequently reported were in the classic form of osteitis fibrosa cystica. In 1934, however, Albright, Aub and Bauer[12] pointed out that other forms of the disorder exist and are not rare pathological curiosities but conditions that every practitioner may expect to meet. In a study of 17 cases they described 6 clinical types of the condition and concluded that 'from the few patients thus far examined we are induced to believe that hyperparathyroidism will turn out to be a fairly common cause of urinary stone and that in future the case in which there is a stone and no bone disease will be the commoner type of hyperparathyroidism'. As knowledge of the disorder advanced evidence accumulated indicating the importance of the condition to the urologist, and in 1941 Albright[13] confirmed his earlier opinion by reporting that symptoms associated with nephrolithiasis are the commonest first manifestation of hyperparathyroidism. Later reports have fully substantiated this statement. At the Mayo Clinic 15-20 cases of hyperparathyroidism are diagnosed annually and, according to Black[14] (1953), these can be 'attributed almost entirely to a more careful screening of patients with urinary lithiasis for hypercalcaemia'.

In tracing the history of the condition it is both interesting and important to observe how hyperparathyroidism, initially described as a bone disease, has progressed into the sphere of the urologist. A growing proportion of cases are being diagnosed because of renal changes alone and the careful investigation of all cases of renal calculus attending urological clinics is becoming increasingly rewarding in the early diagnosis of hyperparathyroidism.

THE PARATHYROID GLANDS

Anatomy

The parathyroid glands are two pairs of reddish-brown glands each about the size of a small pea and ellipsoid in shape. They are situated usually in relation to the posterior aspect of the thyroid lobes.

The superior gland is fairly constant in position, lying behind the upper third of the lobe and related to the lateral surface of the trachea. It is stated to be invariably between the true capsule of the gland and its fascial sheath.

The inferior gland is situated usually behind the lower part of the lobe, either above or below the inferior thyroid artery as it enters the thyroid substance. Considerable variations, however, exist. The gland may be found behind the oesophagus, either in the neck or in the posterior mediastium, or it may be in the retrosternal space Occasionally it may be situated within the thyroid substance.

The cells are of two types: (1) Chief or non-granular cells with faintly staining nuclei and (2) a few eosinophil cells with well-marked nuclei.

Physiology

The active principle of the parathyroids is parathormone, first isolated by Collip[15] in 1925. The most striking and obvious effect of parathormone is on the level of serum calcium. In health the total serum calcium is between 9 and 11 mg.%. This consists of (*a*) non-diffusible calcium bound to protein, usually 4-5 mg.% and fluctuating according to the level of the serum proteins (McLean and Hastings,[16]) (*b*) calcium ions which are freely diffusible and usually total 4·75-6·25 mg.%, and (*c*) a small unionized but diffusible quantity (0·25 mg.%) of calcium in the form of organic salts such as calcium citrate. The solubility product of calcium and phosphate ions in the serum is constant and this means that a rise in calcium ions leads to a fall in phosphate ions and vice versa. Normally the serum is physically supersaturated with both these ions. This mechanism is controlled by parathormone.

HYPERPARATHYROIDISM

Clinical hyperparathyroidism may result from a localized tumour (adenoma or carcinoma) or diffuse hyperplasia of all the parathyroid glands. The lesion most often causing primary hyperparathyroidism is a single adenoma of one of the glands. This occurred in 84% of the cases described by Hellström[17] in 1954 and in 87% of the cases in Black and Zinner's 1956 series.[18] Adenomas are equally frequent on the right and left sides and are 5 times more common in the lower than the upper glands (Norris[19]). The same observer, in his review of 322 cases in the world literature up to 1947, found that 90% of adenomas are in the upper or lower glands and 10% are aberrant; of the aberrant tumours 63% are mediastinal, 30% within the thyroid gland and 7% behind the oesophagus.

Hellström,[17] in 39 adenomata, found one case with 2 glands involved; 14% of his cases showed primary hyperplasia. Black and Zinner[18] report that primary hyperplasia was found in 10% of their series of 207 cases and when seen it was recognized by enlargement of all the parathyroid glands, the hyperplastic tissue being darker in colour and more lobulated than the usual adenoma. They observed an unusual polyendocrine syndrome in 5 cases, with associated hyperinsulinism and hyperparathyroidism, and (in 3 of the 5) a pituitary tumour also. In this series more than one parathyroid was always involved, and under some circumstances all the glands were involved. Carcinomas of the parathyroid are rare; Gentile *et al.*[20] collected 21 examples from

the literature and Black *et al.*[18] reported an incidence of between 0·5 and 1·0% in their series.

The adenomata are usually well circumscribed, brownish-red in colour and soft in consistency. They vary in diameter from 1 to 3 cm. There is no constant histological pattern. They may be composed of (1) small or large 'chief' cells, which are larger than the corresponding cells of the parathyroid, (2) large water-clear cells, or (3) oxyphil cells. It is common to find a mixture of all cell types.

Hypertrophy of all four glands is usually characterized by a proliferation of water-clear cells only. These are larger than normal, with peripherally placed nuclei. Usually no oxyphil or chief cells can be found.

An overproduction of parathormone by any of the above will produce abnormal physiological and biochemical changes. Briefly these are:

1. Elevation of serum calcium.
2. Increased excretion of calcium in the urine.
3. Increased excretion of phosphate in the urine.
4. Decreased serum inorganic phosphorus.
5. Increase in the serum alkaline phosphatase of the blood.

There are two main theories on the mode of action of the excess of parathormone. Thompson and Collip [21] believe that parathormone stimulates osteoclastic activity which leads to widespread lacunar absorption, the liberated calcium and phosphate being absorbed into the circulation, and the excess being excreted by the kidneys. Jahan and Pitts[22] carried out animal experiments to test this theory and state that the hypercalcaemia (and resultant hypercalciuria) produced by the excess of parathormone are dependent on its extrarenal actions of mobilizing calcium from the body stores and not on any specific depression of renal tubular reabsorption of either calcium or phosphorus. Milne[23] after experimental observations on human calcium metabolism supports this theory and further experimental work to substantiate it has been done by Stewart and Bowen[24] and Talmage *et al.*[25] The second theory is that of Albright,[26] who states that the excess of parathormone affects the renal threshold of serum phosphates so that an excess is excreted in the urine. The fall in serum-phosphate ions results in mobilization of calcium from the bones in order to maintain the solubility product at a constant level. The serum calcium is thus raised and any excess is excreted in the urine.

The bone lesions are the result of decalcification. Renal changes occur in almost every case, but the cause is as yet not established. There appears to be structural or functional tubular damage resulting either from increased excretion of calcium and phosphorus, or a toxic process *via* the parathyroid hormone, or both these factors. Parathormone produces increased serum calcium, but increased serum calcium does not necessarily induce nephrocalcinosis. Engel[27] suggests that parathormone depolymerizes ground substance and thereby increases calcifiability; Baker *et al.*[28] carried out a series of experiments on rats and produced evidence to support this view. The resulting renal changes are interstitial fibrosis, calcification and infiltration by lymphocytes and plasma cells, cystic tubular dilatation, and sometimes calcification of tubular basement membranes and relative absence of active glomerulitis or involvement of tubular epithelial cells. The interstitial calcium deposition is mainly peritubular and is preceded by damage inflicted on kidney tissue. Further damage results from interference with tubular function and tubular obstruction (Anderson[29]).

Clinical Features

Having due regard to the physiological, biochemical and pathological changes it can be seen that the symptoms and signs of hyperparathyroidism can be distributed under three sub-headings:

1. *General.* General symptoms such as lassitude, muscle weakness, nausea or vomiting, and constipation may occur. They have been attributed to the hypercalcaemia (Morele[30]). Various other symptoms have been reported. Hunter and Turnbull[31] noted a diminution in auditory acuity and Simpson[32] reported 5 cases of deafness and 1 of aphonia. Bellin and Gerswhin[33] noted calcification in the ear drums in a patient with hyperparathyroidism. Cogan[34] described calcification in the superficial layers of the periphery of the cornea leading to 'band keratitis'. Fitz and Hallman[35] report the occasional occurrence of psychoses associated with hyperparathyroidism. Cases of intense hypercalcaemia and 'parathyroid poisoning' have been reported (Dawson and Struthers[36]), there being gross metastatic deposit of calcium in almost every organ.

2. *Due to Bone Disease.* Early symptoms of skeletal involvement are bone pain and tenderness. General decalcification of the skeleton eventually leads to deformities and pathological fractures; bone cysts and tumours also appear. Tumours and cysts arise most commonly at the ends of the long bones and in the metacarpals and metatarsals, and bone tumour may be the presenting symptom. They may also occur in the jaw, so that the patient may first present with an epulis. Early bone changes which may be evident on X-ray examination are a 'ground-glass' appearance of the vault of the skull, subperiostial bone absorption in the phalanges of the hands, particularly the middle phalanges, and loss of the lamina dura of the teeth. The bony changes of hyperparathyroidism must be differentiated from those of osteoporosis, osteomalacia, Paget's disease, polyostotic fibrous dysplasia, multiple myeloma, metastatic osseous malignancy. Less common conditions which have at times been confused are xanthomatosis, Gaucher's disease, and benign metastasizing haemangioma.

3. *Due to Renal Disease.* Polyuria and polydipsia, without radiological changes in the kidneys, are often marked clinical features of hyperparathyroidism. These are thought to be due to the increased excretion of calcium and phosphate. Renal colic, parenchymal renal calcification, nephrocalcinosis and renal stone commonly occur.

In 1947 Norris[19] reported that out of a total of 322 cases reviewed by him, only 17 showed nephrocalcinosis or renal stone alone (5%). In 1953 Lahey and Murphy[37] reported 29 cases of hyperparathyroidism of which 4 presented with only nephrocalcinosis or renal stone (14%), Richardson[38] reported 11 cases of which 4 presented with nephrocalcinosis or renal stone alone (36%), and Black[14] reported 112 cases of hyperparathyroidism of which 73 showed only nephrocalcinosis or renal stone (65%). In 1954 Hellström[17] reported 50 cases of which 27 showed only nephrocalcinosis or renal stone (54%). In the latest published series, of 207 proven cases of hyperparathyroidism to 1954 (Black and Zinner[18]), 80% had renal stone or nephrocalcinosis.

It is thus evident that a growing proportion of cases of hyperparathyroidism have been diagnosed because of renal changes alone. These cases have been detected by the careful investigation of renal calculus cases attending urological clinics. It is thus an obligatory routine to test for hyperparathyroidism in every case of nephrocalcinosis or renal stone and experience has shown that this routine must not be confined to multiple, bilateral or recurrent stones.

The percentage of all cases of renal stone which are due to hyperparathyroidism is unknown. Cook and Keating,[39] in 1945, reported that out of 850 cases of renal stone 1·65% had proven hyperparathyroidism. Cope,[40] in 1942, suggested that 10-15% of cases of renal stone eventually showed hyperparathyroidism. Black and Zinner[18] now agree with a possible figure of 5% incidence of hyperparathyroidism among patients having renal stone. Although the commonest cause of nephrocalcinosis (Fig. 1) is hyperparathyroidism, it may also occur in hyperchloraemic acidosis, chronic pyelonephritis, chronic glomerulonephritis,[41] vitamin-D intoxication, milk-alkali (Burnett's) syndrome, sarcoidosis and 'idiopathic' hypercalciuria.

Diagnosis

The final diagnosis of hyperparathyroidism once suspected is made on the biochemical findings. The early and frequent occurrence of renal lesions alone has already been stressed, as has the necessity for full investigation of all cases of renal stone or nephrocalcinosis. The following investigations must be carried out.

1. *Serum Calcium.* The presence of a raised serum calcium is the most important single finding. Several estimations should be carried out at intervals, and repeated over a long period if necessary once the condition is suspected. The readings should be consistent and high figures present no difficulty in diagnosis. Ideally the same biochemist should carry out the estimations and his standard top normal figure should be well established if small rises are also to become of diagnostic value. The serum protein should be estimated simultaneously, since if it is low the serum calcium reading may be normal or low and may conceal an elevated ionic calcium. The chart provided by McClean and Hastings[16] is of value in arriving at the correct figure for the ionic calcium (Fig. 2).

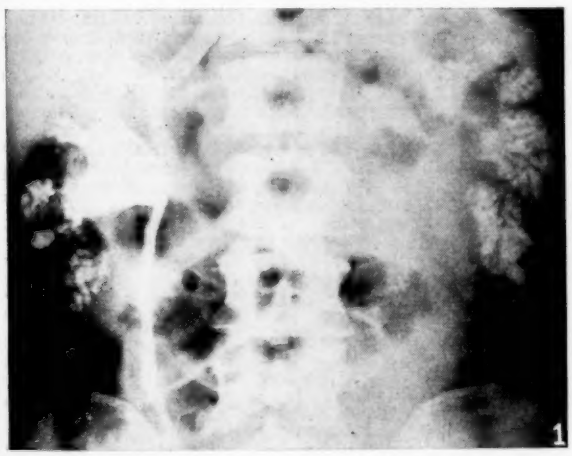

Fig. 1. Nephrocalcinosis.

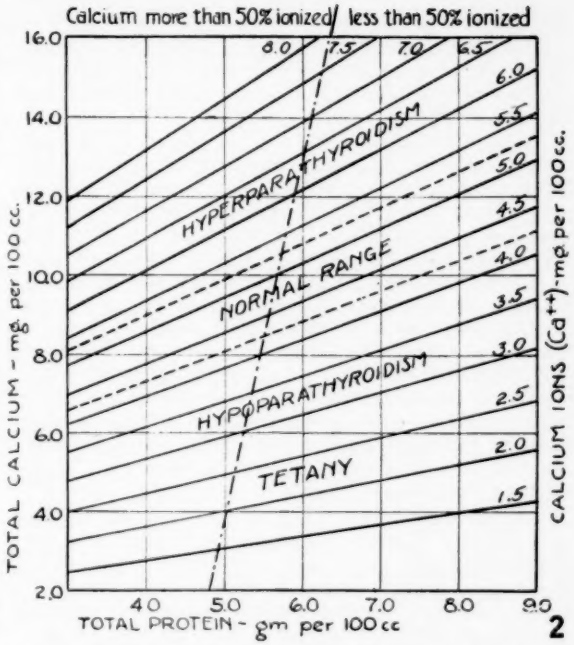

Fig. 2. Chart for calculation of Ca^{++} concentration from total protein and total calcium of serum or plasma (McClean and Hastings,[16] by permission of the American Journal of the Medical Sciences).

2. *Serum Phosphorus.* A low figure for the phosphorus content of the blood, along with a high serum calcium, is strongly suggestive of hyperparathyroidism. A normal or raised figure does not however rule out the possibility, for renal damage may lead to phosphorus retention.

3. *The 24-hour Urinary Calcium.* Figures for the urinary calcium show wide variations. A finding of hypercalciuria on a low calcium diet favours a diagnosis of hyperparathyroidism, but in more than half the cases examined by Hellström[17] the urinary calcium was within normal limits. Hypercalciuria also occurs in many other conditions.[42]

4. *Serum Alkaline Phosphatase.* This is raised only if there are skeletal changes, and such a finding should stimulate a careful radiological search of the skeleton. Early bone changes are found in the skull, the phalanges of the hands and the teeth (as mentioned in a previous paragraph) and these sites should be examined first.

Hypercalcaemia thus emerges as the most important diagnostic feature. It has also been described in (1) metastatic osseous malignancy, (2) multiple myeloma, (3) sarcoid, (4) the milk-alkali (Burnett's) syndrome and (5) vitamin-D intoxication; conditions (1), (2) and (3) have bone lesions which are occasionally difficult to differentiate from those of hyperparathyroidism, and conditions (3), (4) and (5) may present with nephrocalcinosis or renal stone.

Differential Diagnosis

Metastatic Osseous Malignancy. Malignant secondary invasion of bone may give rise to hypercalcaemia. A primary source should be sought for in the common sites, viz. breast, prostate, kidneys, bronchus and thyroid.

Multiple Myeloma. The serum phosphorus is usually normal or raised in multiple myelomatosis, but Snapper[42] reported a case with a low serum phosphorus. Renal damage may lead to phosphorus retention in hyperparathyroidism. Serum phosphorus readings may thus be of little help in the differential diagnosis. Bence-Jones protein has been found in the urine in less than half of the cases of myeloma and when it occurs it may be continuous or periodic, and in scanty or large amounts. Bence-Jones proteinuria may also occur in myxoedema, leukaemia and carcinoma. The serum alkaline phosphatase, however, is usually normal in multiple myeloma. An increase in the plasma proteins, particularly affecting the globulins, is characteristically found in many cases of multiple myelomatosis. A bone-marrow biopsy with the demonstration of an increased number of plasma cells would confirm the diagnosis.

Sarcoid. Bone lesions, when present, are in the nature of circumscribed lesions of coarse trabeculation and sharply punched out small cyst-like areas, chiefly in the hands and feet. Harrel and Fisher[43] found that hypercalcaemia was not confined to cases with bone disease. Difficulty is mainly encountered in the cases in which there are only metabolic changes and there is an obscure hypercalcaemia. Serum phosphorus estimations may be equivocal. Dent's cortisone test[44] is a valuable diagnostic aid in both this condition and *vitamin-D intoxication.* In this test 150 mg. of cortisone are given for 10 days and the serum calcium is estimated on the 5th, 8th and 10th days. The hypercalcaemia of sarcoidosis will rapidly fall, usually to normal levels, during this time, as will that of vitamin-D intoxication.

Milk-alkali (Burnett) Syndrome.[45] In this condition there is a history of prolonged and excessive intake of milk and absorbable alkali. There is hypercalcaemia without hypercalciuria or hypophosphataemia. The serum alkaline phosphatase is normal and there is a mild alkalosis. Calcinosis is present, manifested especially by an ocular lesion resembling band keratitis. The history is the most important factor in the differential diagnosis. After withdrawal of milk and alkali powders an improvement in the clinical state results and the hypercalcaemia often diminishes.

Treatment

Surgical removal of the affected gland or glands will normally bring about a complete cure of hyperparathyroidism. Exploration of the neck is indicated when the diagnosis of hyperparathyroidism has been confirmed by the biochemical findings. It is highly unlikely that any swelling of the affected glands can be detected on clinical examination. It is rarely necessary to do more than the standard neck dissection with careful identification of the parathyroid glands. Methodical identification of each of the four glands is the method of choice. If no tumour is found in the normal situations of the glands, the search should be extended into the upper part of the thorax, splitting the sternum if necessary The retrosternal and retro-oesophageal spaces should both be explored and a branch of the inferior thyroid artery running downwards into the thorax may point the way to a parathyroid tumour. Rarely the affected parathyroid may be buried within the thyroid substance. If exploration of the thyroid confirms this, the tumour may be enucleated or subtotal thyroidectomy performed. In the absence of a tumour, if demonstrable hyperplasia of all the glands is found three should be resected.

After operation an immediate fall occurs in the urinary calcium and phosphorus excretion. The level of calcium in the serum falls to normal within 1-4 days. When bone disease is present operation is sometimes followed by a fall of serum calcium to sub-normal levels as the decalcified bones absorb calcium from the plasma, the remaining parathyroid tissue being insufficient to prevent this reversal. In some cases the fall of the serum calcium to subnormal limits progresses to the point of tetany. Parenteral administration of calcium salts is required. It can be given either intramuscularly or intravenously, usually in the form of the gluconate. Parathyroid hormone and calciferol or dihydrotachysterol, used effectively and logically in tetany which follows damage to the parathyroid glands in the course of thyroidectomy, are relatively ineffective in this condition.

SUMMARY

The history of primary hyperparathyroidism is presented. Initially described in its classic form of osteitis fibrosa cystica only, its gradual recognition as a disease of major importance to the urologist is outlined.

The anatomy, physiology and pathology of the parathyroid glands and the clinical features, diagnosis and treatment of primary hyperparathyroidism are discussed.

The high proportion of patients with hyperparathyroidism presenting with renal changes alone is emphasized.

The careful investigation of all patients with nephrocalcinosis or renal stone for hyperparathyroidism is stressed as an obligatory routine—not only in cases where the stones are multiple, bilateral or recurrent.

Hypercalcaemia is considered to be the most important single diagnostic feature. Its possible causes are discussed.

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THE ROLE OF THE MEDICAL MAN IN THE PRACTICE OF MANAGEMENT*

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Dr. Serebro referred to the great industrial development that had taken place in South Africa in recent years, and stated that in 1953, 775,000 people of all races were employed in secondary industry alone,[1] with an annual wage bill of £445 million[2] and an industrial output to the value of £1,140 million[1] (exclusive of the mining and agricultural industries, and the activities of central and local government, who were employers of labour on a vast scale). He said the aftermath of World War II had seen a gradual normalization in the industrial picture, and that competition both from within and from overseas was making itself felt. With this came the realization that an apparently cheap and plentiful labour supply was in reality not so cheap and in the main was unskilled. He referred also to other weaknesses in the supportive and para-industrial structure, and mentioned particularly defects in the educational system from the industrial point of view.[3] Dr. Serebro spoke of the necessity of conserving man-power and using it to the best advantage,[5] and went on to say:

We are beginning to understand the methods whereby the best use can be made of our manpower. This is possible by the application of management techniques, where the ultimate targets are efficiency and productivity, acquired by methods which do not produce physical or mental fatigue or frustration in our workers. This goal is reached by a management which has the knowledge and the skills necessary to achieve its purpose, without recourse to the blood-sweat-and-tears or the carrot-on-a-stick techniques. The work of Elton Mayo[6] in his experiments at Hawthorne with the Western Electric Company together with his earlier work in a mule-spinning department of a textile mill are classic examples of what management can achieve if it is ready to apply itself to the various industrial problems with human relations as the starting point.[7] For even with the best tools, with high-grade equipment, good working conditions and reasonably skilled workers, the results at times, if measured even in terms of efficiency and productivity alone, are sometimes most disappointing. This applies not only to the factory, but to any undertaking whether private or public, under central or local government, as well as hospitals, clinics, laboratories and even educational institutions. Such failure is an indication of defective management.

The care and the maintenance of the health of the individual, in both the preventative and the therapeutic aspects, is often referred to as personal health. This personal health is the business of the medical man in general practice. The care and maintenance of optimum working conditions, such as lighting, ventilation, working comfort, environmental humidity and temperature, together with personal health, is known as occupational health, and is the concern of the medical man in industry. However, it is not enough to maintain occupational health alone; for today the doctor in industry must solve problems of a more complex nature concerned with inter-personal behaviour. Among these, priority is given to the understanding and the practice of active human relations which facilitate communication, and assists in worker co-operation, resulting in such a low rate of sickness absenteeism and improved or increased productivity. These results are an indication of efficient management.

The medical man in industry is the person concerned with the sickness absenteeism figures. He it is who, in matters relative to occupational health, is the arbiter between the worker and management. He is in a privileged and unique position from which he is able to judge whether management is shepherding its industrial flock smoothly and without hitches. The medical man does not gauge only with instruments of glass, steel or mercury, but rather by his skilled observation of human behaviour under specific conditions. These are reflected in his medical certificates. Using this type of gauge, the medical man can watch the fluctuations in the sickness rate, and decide to what degree it is real or apparent. The doctor cannot usually stem an apparent or artificial absenteeism rate. He cannot compete against nor diagnose from mere symptoms. It is essential to realize that a rising rate of sickness absenteeism is not necessarily indicative of an impaired standard of medicine or bad doctoring, but commonly points to some inherent weakness in labour-management relationship. Management chooses its labour force and creates the working environment; hence the responsibility for any failure rests entirely with management. Apart from certification, the medical man is capable of pointing out to management stresses and faults in its other industrial relations and environment and giving relative advice.[8]

The doctor in industry is in contact with workers all the time and becomes aware of their likes and dislikes in relation to management. With discrimination he can sort the facts from the fantasies, and again can give management definite indications of structural managerial defects. 'The actions of the medical man in industry, his decisions and his responsibilities are far-reaching, and have management implications.'[9]

During the last two decades, management has developed practical techniques based on knowledge derived from economics, mathematics, psychology and sociology. To develop these techniques it drew from the fundamental as well as from the applied sciences, and carried out experiments which were not always

* A paper read at a combined meeting of the South African Society of Occupational Health (a Group of the Medical Association of South Africa) and the National Development Foundation of South Africa, Durban, 20 September 1957.

successful. Yet, out of these successes and failures, with the experience of the years, new concepts arose and principles were elaborated which today constitute the standards of management.

In the welter of a multiplicity of techniques, well-tried procedures have been arrived at for getting things done. Management made this possible by its reliance on expert guidance by the statistician, the engineer, the industrial psychologist and the works accountant. Through all this progress there has been one dominant difficulty, and that lay in the field of human behaviour. To help unravel the complexities of human nature was needed an expert in human structure and function, with his understanding of all the phases of man from babyhood to old age. Thus the medical man takes his place, with those other experts who can advise management in the industrial field.

Medicine, like Management, has throughout its existence borrowed from the arts and appropriated from the sciences. It has modified, adapted and used methods and techniques and equipment from the chemist, the physicist, and recently the atomic technologist. Management experts from the Old World and New World as well as those of this country have already entered the arena of improved human relations, and have staked their claims in this field. Here we find other professional men intimately concerned with good human relations,[10] who invite the cooperation of the medical man in this field. Medical men who are interested in these activities are invited to participate in some measure of inter-professional education which will assist them in dove-tailing with their fellow experts in the Management team.

As medical men, by reason of our training and outlook, we have a direct interest in human beings, and it is right and proper for those of us who are connected with industrial organizations to assist management by taking an active part in the personnel-management aspect of those undertakings; and at the same time to take part in the inter-professional exchange on common and yet overlapping matters. This will assist the doctor in Industry in acquiring the knowledge and the necessary tools which are essential for his own work as well as for the integration of his orbit in the wider sphere of Management.

There is already in existence an ideal forum in this country in the form of numerous management and supervisory meetings which take place throughout the year. These are organized by such bodies as the National Development Foundation, the Institute of Personnel Management and the Institute of Production Engineers. It is at this level that the doctor in Industry can make inter-professional contact on a worth-while and practical basis.

Finally, the industrial management experts are in contact with all types of people connected with Industry, and are fully involved in the problems of human relations. It therefore seems logical to assume that medical men in industry and the management experts have a common purpose. There is scope for a close liaison and symbiosis, for the formation of a team that will have as its main object the furtherance of good Labour-Management relations of real value to Industry.

SUMMARY

South Africa has developed her secondary industry, and as an industrial producer must compete in the various commercial markets. In order to do this it must have a skilled labour force, which in the main is lacking. Management techniques assist in the conservation of our existing manpower.

The medical man in industry must not only concern himself with personal and occupational health problems, but must solve problems of inter-human behaviour. The doctor can assist management in its functions as well as indicating to management weaknesses in itself and in its programmes, by the mechanism of medical certification and sorted opinions of the workers. These opinions can give management indications of managerial defects.

Management experts are already active in the field of improved human relations, but invite the co-operation of the medical man in this field as well as in the aspect of inter-professional education.

There already exists a forum in this country in the form of management and supervisory meetings, which are held throughout the year, organised by various bodies interested in personnel management. On a practical basis the medical man in industry can make the necessary contacts and acquire the knowledge that will equip him further, and make him the appropriate person to take an active place in the management team.

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THE MEDICAL PROFESSION IN THE SERVICE OF SOUTH AFRICA'S PRODUCTIVE ENTERPRISE*

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In the early days of the Industrial Revolution, the Industrialist is reputed to have cared little for the welfare of his employees. Today, however, he has a highly developed social conscience and is, in any case, forced by the general shortage of labour and the high cost of training to take an interest in the health and welfare of his employees. Nowadays, even if labour were in free supply, the employer must face the cost of training each replacement to the high degree of speed, efficiency and skill required in this competitive world. Problems of absenteeism and labour turnover, aggravated by the labour shortage and specialised skills, have become of the most major importance to the very existence of a producing concern.

TABLE I. GROWTH OF THE INDUSTRIAL POPULATION

Year	*Number of Workers*		*Index Base:* 1924—100	
	White	*Non-White*	*White*	*Non-White*
1924	53,450	99,297	100·0	100·0
1932	68,981	95,809	129·0	95·5
1939	115,292	199,196	215·7	200·6
1944	133,518	297,884	249·8	299·9
1949	191,291	426,888	357·9	429·9
1952	217,447	523,865	406·8	527·6

Table I illustrates the increase in the number of industrial employees during the 28 years preceding 1952. This increase has continued during the past 5 years and the shortage of labour, particularly of skilled labour, continues to be chronic.

* A paper read at a combined meeting of the South African Society of Occupational Health (a group of the Medical Association of South Africa) and the National Development Foundation of South Africa, Durban.

ANALYSIS OF MEDICAL SERVICES TO INDUSTRY

Analysed from the administrative/financial angle there are, generally speaking, 3 main types of medical schemes in the service of industry:

1. *Factory Medical Care*

Provision of first aid facilities at the works has been required since the earliest industrial legislation was introduced. Today larger firms have developed these first-aid facilities to a stage where a full-time nurse is employed and the services of a part-time doctor are retained to provide a reasonably comprehensive 'curative' service. The part-time doctor not only attends to accident injuries but also to minor illnesses. Employees can therefore have their minor ailments treated at work and so avoid having to take time off and lose pay to go and visit a doctor. Large concerns in the country districts or in smaller centres have gone so far as to erect a sick bay or small hospital with a nursing staff and perhaps a full-time doctor.

A more recent development has been the provision of a factory medical service run on 'preventive' rather than on 'curative' lines. From the point of view of efficient production, the employer is concerned with the promotion and maintenance of the fitness and health of his employees. Except to achieve a speedy return to work and from a general welfare point of view, productive efficiency is not affected by the arrangements made for the medical care of employees who are absent because of illness. The 'preventive' service therefore aims at maintaining a continuing interest in the employees' health and welfare in an attempt to foresee and avoid illness and sick absenteeism and to promote the employees' efficiency and happiness in his work. This introduces a number of new factors into the field of the factory medical service. A man's attitude to, and efficiency at, his work is influenced by the health of his family as well as by his own health, and psychological aspects are almost as important as the potential spread of disease. Further, the factory doctor becomes an important consultant of Management on safety measures, the effect of certain types of work on health, and in almost all welfare matters. In his privileged position of being in possession of confidential personal details concerning the employee, his advice is of inestimable value in dealing with personnel problems.

If the factory doctor's advice is to be acceptable to Management, however, his approach to the job must be tuned to the requirements of industry just as much as that of the other department managers in the factory. The costs of the medical department are every bit as important as the costs of the engineering and maintenance departments or, for that matter, any of the production costs. The industrial Wage Bill has already soared to unprecedented heights and any increased charges must prove their value by improving production or they will merely add to the cost of the product and further increase the vicious circle of the rising cost of living.

2. *Medical Benefit Funds*

The medical service at the factory, whether merely 'curative' in nature or a more pretentious 'preventive' scheme, has often been combined with a scheme for the provision of sick pay for employees financed through the establishment of a medical benefit fund. The provision of medical services at the factory will tend to reduce sick absenteeism, and the necessity of obtaining a certificate from the factory doctor to qualify for sick pay will offset the new incentive for the employee to absent himself from work on medical grounds.

The benefit fund is financed normally by equal contributions from employer and employees. Its two most important aspects are, firstly, the provision of medical services to employees at a known cost (being the amount of their contributions to the fund), provided that the fund operates within its financial resources and, secondly, that it takes over from the employer the legal obligation to allow up to 12 days sick leave per annum and pays its members sick pay in the event of illness on more generous terms and for longer periods than required by law.

From the employees' point of view, therefore, he insures himself, by the payment of contributions to the Fund, against irregular and unknown medical expenses and the possibility of a cessation of income due to illness. According to its financial resources, the fund may or may not cover specialists and hospital expenses. If it does, a further important financial burden is lifted from the employees' shoulders.

From the employer's point of view, always provided that the fund operates within its financial resources, the costs of the medical service are known and limited to the amount of his contributions to the fund and the welfare of employees is improved by the introduction of more generous sick pay provisions.

Benefit funds often increase the scope of their medical service by appointing a panel of doctors, instead of one factory doctor, so as to allow employees some freedom of choice of their medical practitioner. Unless the panel is very close-knit, however, a 'preventive' medical service becomes impossible and, except so far as general welfare is concerned, the usefulness of the scheme to factory management disappears because the intimate co-operation between the factory doctor and factory management on employee affairs no longer exists. The medical service ceases to be a department of the factory tuned to improving and reducing the cost of the product and becomes merely an additional charge to general welfare, being provided for goodwill purposes only. Where a benefit fund appoints a panel of doctors in addition to a factory doctor, it would be wise to permit only the factory doctor to issue certificates for payment of sick-pay. If panel doctors are permitted to authorize paid sick leave, that doctor who is most generous in this way will be the most popular amongst the employee members and panel doctors might inadvertently earn the reputation of competing for patients by unnecessary issue of sick-pay certificates.

Unfortunately benefit funds must, with normal financial prodence, place limitations on the benefits available to members in order to remain solvent. Sick-pay is therefore usually limited to slightly less than normal pay for the first 2-4 weeks of an illness and thereafter is paid on a reducing scale for a maximum of usually 3 months in any one year. Chronic illness is also normally excluded from benefit and, to prevent persons from joining the fund to obtain expensive treatment of existing ailments and then moving elsewhere, illnesses which pre-date membership of the fund are excluded. Certain medical expenditures are often regarded as avoidable or not essential to basic health and therefore not a reasonable charge to the fund; so that expenditures connected with maternity, treatment for sterility and cosmetic effect, V.D. and others are very often left to the individual to meet himself.

3. *Medical Aid Societies*

A medical aid society is a straight-forward insurance scheme. It is a two-way insurance: the medical practitioner is guaranteed against bad debts because the society undertakes to pay his accounts, and the member is insured against heavy medical expenditure.

The essential differences between a benefit fund and a medical aid society are that the society does not provide sick pay, nor does it employ a medical staff (except, of course, a medical adviser who interprets accounts received from medical practitioners and translates the nature of the illness so that the lay committee may apply the society's rules). Further, at the insistence of the Medical Association, a medical aid society incorporates the principle of free choice of doctor (the members may select any medical practitioner to attend them) and there is no question of a panel of doctors.

All trace of control by the society over the medical practitioners providing services for their members is therefore eliminated. Medical aid societies are fortunate that an extremely high standard of professional integrity is the general rule in medical practice, because a society's funds could be milked in a very short space of time if doctors providing services for its members practised over-visiting and other money-making methods. Against these possibilities the society has only two safeguards: firstly, a contract with the Medical Association laying down a tariff of maximum fees for all possible medical procedures—the elimination of bad debts enables the medical profession to offer lower fees than normally charged in private practice—and secondly, by requiring the member to pay a portion of the accounts himself. This second safeguard has its limitations, because the normal insurance practice of requiring the insured to pay the first £5 (or whatever the relevant sum would be) serves no purpose. The society therefore fixes a proportion of the account which it will carry itself and requires the member to refund the balance. The society then hopes that should a medical practitioner indulge in over-visiting or some other

malpractice, the member will find himself a new doctor. There is however a flaw in this latter safeguard because if a doctor should visit him more frequently than is strictly necessary, the member's ego is probably inflated by the doctor's apparent concern for his welfare.

The problem of collecting that portion of the medical accounts refundable by members (and ex-members who obtained treatment immediately before withdrawing from the society) is one of the major difficulties facing any society which tries to adopt this method of indirectly controlling medical expenses. However, there are other reasons for requiring a member to make some contribution towards his actual expenses; namely, that few societies can afford to provide full cover and, further, the member is likely to be more appreciative of the work done by the medical practitioner and is less likely to abuse his privileges if it directly affects his pocket to some extent. This provision therefore operates in the interests of both the society and the medical practitioner.

The industrial employer need have little to do with the administration of a medical aid society and he makes it possible for his staff to enter such a scheme (or establishes one exclusively for them) in their interests and not his own. It is quite common practice for an employer to pay portion of the employee members' contribution to a medical aid society. The chief attraction of this type of medical cover to an employer is the fixed cost of the subsidy he pays. It should be stressed that an employer's investment in a medical aid society is recurring and merely a provision for treatment of employees' sickness. Having made this contribution the employer may, as a layman, consider that he has gone far enough. In fact he has not made an investment to promote the health of his employees at all; his contribution is a welfare expenditure and an added cost of production and must be regarded in this light.

An industrial employer may, however, establish or join a medical aid society in order to provide the specialist and hospital care not available at the factory medical department.

The medical aid society suffers from the same drawbacks as a benefit fund in having to restrict and limit benefits in order to remain solvent. The Achilles heel of a medical aid society is, however, the absence of control over chargeable services rendered to members. It is not unknown for a medical aid society to be forced to convert the nature of its organization to that of a benefit fund with a panel of doctors in order to avoid insolvency. I do not have to dwell on this point since the information is gleaned from the pages of the *South African Medical Journal* and the Medical Association is already concerned at the implications of such events.

APPROVED FEES FOR MEDICAL PRACTITIONERS

There are 4 bases recognized by the Medical Association for the payment of medical practitioners employed by or cooperating with one of the abovementioned schemes:

(*a*) As a full-time employee the doctor is paid a salary.

(*b*) As a part-time employee the doctor is paid at the rate of £2 2s. 0d. per hour.

(*c*) Doctors on the panel of a benefit fund may be paid £1 2s. 6d. per annum per European and 12s. 0d. per annum per non-European registered on their panel, or

(*d*) Panel doctors or doctors treating members of approved medical aid societies are paid a fee based on services rendered and calculated in accordance with a tariff of fees drawn up by the Medical Association for all possible medical procedures.

The complexities of completing an insurance claim (i.e. the medical aid society's claim form) so that accounts are presented in a form understandable by the lay committee of the society, are often irksome to the medical practitioner and, where incorrectly completed, lead to considerable delays in payment by the society. Whilst most doctors complete these insurance forms correctly, others, probably owing to lack of interest or lack of explanation, do not. This gives rise to delays which interrupt the whole process of approval and payment by the society.

On the other hand, the *per capita* fees allowed for panel doctors of a benefit fund are perhaps too low to provide any incentive for the doctor to pay anything more than a cursory interest in the general health and well-being of the member patients on his panel. In fact the panel doctor is intended only to provide a 'curative' service. An interesting illustration has been given by the British National Health Service where, it is reported, general practitioners have complained that the scheme does not allow the time or the remuneration for patients who demand a 'check over'.

ADMINISTRATION EXPENSES

The proportion of revenue absorbed in administering medical aid societies and benefit funds has an important bearing on the finances available for the provision of benefits. The marginal cost of administering a benefit fund for factory employees may be so small as to be disregarded. The work connected with the fund's accounts can be quite easily undertaken by the existing factory accounts clerks and the payment of sick-pay can be absorbed into the pay-office routine without an increase in staff. If the fund is administered by a committee, the committee members are normally appointed or elected in an honorary capacity. The marginal cost of administering the scheme might therefore be merely the few shillings spent on additional stationery.

A medical aid society on the other hand must face heavy costs of administration; according to the size of the scheme, these may absorb between 20% and 35% of the society's revenue from contributions. A society must have at least 2,000 members in order to achieve a reasonable spread of the insurance risk. It follows that few concerns are of sufficient size to establish an internal society without the risks being unduly concentrated. Medical aid societies are therefore often established to cover a group of smaller concerns, and the maintenance of membership and contribution records becomes more complex and probably a full-time job for one or more clerks. A trained and experienced staff is required to check and assess the benefits on claim forms received, and a complex system of controlling benefits to within the maximum limitations imposed by the rules must be instituted. The sums of money invested in office equipment are sometimes very large. Payments must be made to a wide circle of medical practitioners and the services of a medical referee (whose duty is not the provision of benefits) must be retained. The work done by the committee may reach proportions where it becomes necessary for the society to pay fees to committee members.

A very large society, spreading administration costs over a large number of contributors, may be able to operate on 20% of its income, but the ordinary society can be congratulated on its efficiency if it manages to keep its administration costs below 30% of its contribution income.

Other Medical Schemes

I have for convenience grouped all medical schemes under 3 headings (see above) namely: (*a*) The factory medical service financed directly by the employer, which may be regarded as an ordinary service department in the factory; (*b*) the benefit fund financed jointly by employer and employee, and (*c*) the medical aid society financed by premiums paid by the employee (normally subsidized by the employer).

There are of course a variety of other schemes but most of them contain the basic features of one or other of these three or a combination of them. One of the societies with which I have been associated, supplements the services provided by the factory medical department, and bears part of the retaining fee paid to the factory doctor, a portion of the cost of medicines and the full cost of specialist and hospital care. The society also provides sick-pay privileges and caters for the employees' families. Comprehensive statistical records of all attendances, illnesses, treatments and absenteeism are maintained at the factory surgery. All employees and their families are medically examined on arrival and thereafter once annually and may obtain free medical treatment at the surgery. The scheme is run on 'preventive' lines and detailed medical records are kept for each individual employee and his family.

In order to introduce the 'medical aid' aspect and allow freedom of choice of general practitioner, this society has extended its scheme to provide what it calls its 'domestic medical service'. Any local general practitioner who wishes to cooperate with the society may have his name added to the open panel providing this service. Employees may, as an alternative to obtaining free treatment at the surgery, call in any panel doctor who, in order to keep the factory records complete, keeps the factory doctor informed of all services provided to employees and their families. This is done by routing accounts for services rendered through the factory surgery, where they are checked and the details inserted on the

record cards. For the same reason, all cases recommended by panel doctors for specialist or hospital care are routed through the factory doctor. 50% of the panel doctor's fees are, however, recovered from the employee patient because these fees cost the society more than double what it would have cost had the necessary treatment been obtained at the factory surgery.

Thus this society attempts to combine all the advantages of the 3 main types of scheme listed above. From the date it introduced its domestic medical service this Society has been threatened by financial difficulties. These difficulties do not arise solely from the extension of its scheme to provide a free choice of doctor because there has been an increase in the number of families dependent on the society to carry their medical expenses. Rising hospital and specialist fees have also taken their toll. Nevertheless, an analysis of the society's accounts shows that the introduction of the medical aid aspect has proved to be a major drain on the society's finances.

SUMMARY AND CONCLUSIONS

Industrial medicine—if I may use that phrase to describe the functions of a factory medical department whose objects are to maintain and promote the health and happiness of employees, keep them at work and, as a natural corollary, improve their efficiency—industrial medicine is a relatively new field for exploitation by medical science. It is perhaps not a new approach and has been used before, but its potentialities have not been fully exploited in South Africa. By striking at the root of industry's absentee problems, the medical profession could provide a valuable service to South Africa's productive enterprise by fostering the development of this branch of its activities. It will, however, be necessary to give further thought to the organization of industrial medicine and the complementary benefit funds and medical aid societies.

Firstly, as I have tried to illustrate, the cost factor is the governing element. If industrial medicine is to be a 'healthy' development in itself, its costs, like those of the engineering departments and consulting engineer's fees, must not represent an increase in the cost of production. Its activities must result in, and its cost be absorbed by, improved production. The object must be a lower-priced product through the improved productive efficiency of labour.

Secondly, the importance of industry's absentee rates and the growth of the industrial population indicate that there is a tremendous untapped field for medical practitioners specialized in industrial medicine. Where a factory is not large enough to employ a full-time doctor, a doctor specialized in this type of work will not fail to find several factories demanding the services of a part-time doctor. It is important to these factories that the factory doctor be a specialist, or at least experienced, in this field because he takes charge of a factory department and not an ordinary surgery. An experienced factory doctor who understands the workings of a factory, general personnel problems and the importance of cost and statistical analysis, would earn his £2 2s. 0d. per hour, whereas the family doctor with no experience of factory problems would not.

Thirdly although the Union Health Department has done some research in the field of industrial medicine and has shown that much can be done in this way to alleviate the problems of absenteeism and even labour turn-over, it remains with the profession to prove its value. It follows that a medical practitioner who undertakes to provide services for an industrial medical scheme must understand the nature of the problems involved and re-tune his approach to the needs of productive efficiency. The industrialist too must be educated to the requirements of industrial medicine and be shown that the payment of a subsidy to a medical aid society is not an investment but a goodwill expenditure and an added cost of production.

Finally it becomes apparent that the Medical Association will have to give further thought to its policy of insisting wherever possible on a free choice of doctor. This policy implies apparently that its members may participate in all medical aid schemes (at a fee lower than that charged in private practice), although such work may not fit in with the ordinary practice of any particular member and the medical aid management must employ medical practitioners who may not be interested in promoting the success of the scheme in question. There appears to be some moral obligation on the medical practitioner to undertake medical aid society work at tariff rates, although he may refuse to do so. The medical aid management may not, however, advise its members that it will not be responsible for payment of fees to any particular doctor. This unusual arrangement may not promote amicable relationships between doctors not particularly interested in this type of practice and the scheme's management.

It is normal insurance practice for the insurance company to nominate the party who will provide the required services to the insured. Presumably there is no legal obligation for a medical aid society to do otherwise, except the terms upon which the Medical Association will approve the scheme, and such approval is necessary before the Association permits its members to participate in the scheme and reduce their charges to medical aid rates. Further, it is important to the lay management of a medical scheme to have the support of the Medical Association, and they would in any case wish to cooperate with the Association to the fullest extent. Nevertheless it would seem to the layman that the mutual interest of the profession and medical aid schemes would be better served if more ordinary rules of contract were given a freer play.

Instead of asking that all members of the Medical Association participate in every medical scheme, each scheme's management might be encouraged to establish a panel of doctors for that scheme. Any doctor who is not interested in this type of work or in that particular scheme, need not have his name added to the panel and the scheme's management, after proper advertisement, will select from the applicants a panel of doctors wide enough to provide adequate freedom of choice for members and comprised only of doctors interested in that particular scheme's success. Where the relationship between the scheme and any of its panel doctors is an unhappy one, they can sever their connection without difficulty. A bad scheme will soon fail, because doctors will have their names withdrawn from the panel and the Association can draw the attention of the scheme's management to its deficiencies, and possibly, circularize its members pointing out the difficulties that have been experienced by doctors on that particular panel. A good scheme will succeed through interested cooperation of the panel doctors and scheme management for their mutual advantage. Such an approach would provide a freedom of contract for industrial schemes which they have so far not enjoyed and would go a long way to solving some of the administrative difficulties experienced by medical aid societies and benefit funds.

INDUSTRIAL PERSONNEL MANAGEMENT

At the combined meeting of the Society of Occupational Health and the National Development Foundation at Durban on 20 September 1957, Dr. A. J. Orenstein in the chair, after the reading of the foregoing papers by Dr. B. Serebro and Mr. R. O. Fowler, discussion ensued, which is summarized as follows:

Dr. D. J. Lapping (Durban): The doctor must be integrated into industry so that he can play an active part in industry generally. The doctor should have equal opportunities in the making of decisions, with other members of top management.

Mr. Charles Bedaux (Johannesburg): The medical services of an organization should be integrated into its personnel department. The function of a personnel department is *inter alia* that of a diplomatic liaison. This department should keep detailed information for the use of top management only. He found that the annual medical check-up of workers was an important part of the medical services of an organization, and one which paid for itself handsomely.

Dr. V. R. A. Croly (Port Elizabeth): By reason of his training and wide outlook the doctor in an organization should be the senior man in its combined medical and personnel department.

Dr. L. Blumberg (Cape Town): The medical man in industry must have status on a common and equal footing with senior executives in any organization. Industry in South Africa needs to conserve its man-power. The industrial medical expert should not be concerned with the sick worker directly; this should be the

function of his clinical colleague. He should act as the advisor to management.

Dr. W. L. Gopsill (Durban): The factory medical officer by the correct approach can do a lot towards increasing productivity through creating an environment in the factory which is satisfying to the workers and to management.

Mr. Fleming (Durban): The doctor should play his role as part of the personnel service. He should confine himself to those activities to which he has been trained. He cannot assume the part of another member of the executive team, such as the engineer. A doctor cannot change his identity.

Dr. A. J. Orenstein (Johannesburg), in summing up, said:

There is a clear distinction between the doctor concerned with the therapeutic or the curative aspects of medicine as applied to industry, and the industrial doctor. These two doctors have interests in different spheres of medical care.

The function of the industrial doctor is to keep industry going all the time. The patching up of those who unfortunately fall by the wayside is the work of those medical men concerned with therapy, and is primarily the function of the general practitioners. The industrial doctor must sit at the round table with other executives and advise them on how to keep the human elements in an organization moving smoothly. He must advise on the environment of the worker, and how to keep the worker contented.

This industrial man should know every process in his organization. He should know how the various machines work, and at the same time be aware of their dangers as well as their productive potential. He should be aware of any toxic processes in a plant and understand the various hazards, and in consultation with the chemist and the engineer he should arrive at minimum standards for safety and production.

At one stage industry did not think of the medical man as productive. Today it must change its tune and march with the times. Industry must take advantage particularly of medical knowledge as well as the other sciences, so that it can produce for itself safety services and other amenities at a cost within its own control. This should be done immediately, and industry should take the initiative in these matters.

In order to do this, it follows that the right industrial medical men should be employed by industry at an early stage, so that they will be of immediate help as well as of long-term advantage to industry.

NAUDÉ VERSUS WHITTLE : JUDGMENT ON APPEAL

In the Supreme Court of South Africa (Appellate Division) the case between Willem Johannes Naudé (appellant) and Butler James Whittle (respondent) was heard on 3 December 1957 (*coram* Fagan, C. J., Steyn, Malan JJ. A., Reynolds et Hall, A. JJ. A.) and judgment was delivered on 9 December 1957 as follows:

JUDGMENT

Fagan, C. J.:

This is an appeal from a judgment given by *Sampson, A. J.*, in the Eastern Districts Local Division.* The action was one for damages for alleged defamation.

The appellant, who was the plaintiff in the Court *a quo*, is a medical practitioner and was at all material times the district surgeon for the Franklin and Zwartberg areas of the Mount Currie District.

At the annual general meeting of the Zwartberg Farmers' Association held on 27 October 1955 and attended by about 50 persons, the respondent (so it was alleged in the declaration) said:

'When Mr. George Cullen of the New Amalfi area committed suicide on the night of 13 October 1955, the District Surgeon, Franklin (meaning and referring to the plaintiff) was telephoned at 10.30 p.m. and refused to appear at that hour, although the circumstances were explained.'

The declaration alleged, and the plea admitted, that it is common knowledge—

(*a*) that it is the professional duty of a medical practitioner, when called on to do so, to render to any person within the area where he carries on practice such professional services as he is capable of performing and as might be required of him;

(*b*) that it is the professional duty of a district surgeon, when called upon to do so, to render such professional services as he is capable of performing, and as might be required of him in all cases where a crime might have been committed involving *inter alia* injury to or death of a human being.

By his statement—so the declaration continued—the defendant (now the respondent) meant and was understood to mean:

(*a*) That on 13 October 1955 at about 10.30 p.m., the plaintiff was informed by telephone that a certain Mr. George Cullen had committed suicide and that the full circumstances attendant upon this tragedy were explained to him;

(*b*) That the purpose of the said telephone conversation with the plaintiff was to request him to proceed to the scene of the tragedy and there to give such professional services as he could and was obliged to do in his capacity as medical practitioner, and as the district surgeon for the area concerned;

(*c*) That it was the professional duty of the plaintiff in all the circumstances, which had been made known to him, as a medical practitioner and as the district surgeon, to have proceeded to the scene of the tragedy as soon as possible in order to render such professional assistance and services as were required of him and as he was able to give;

(*d*) That the plaintiff refused to respond to the said request to render such professional services on the ground merely that it was late at night, and that his refusal was therefore without good or sufficient reason or excuse;

(*e*) That, therefore, the plaintiff was guilty of a serious dereliction of his professional duties as a medical practitioner and as a district surgeon;

(*f*) That his conduct aforesaid was, in the circumstances, grossly unprofessional and unethical and not in keeping with the high calling of his profession as a medical practitioner;

(*g*) That his conduct was, in the circumstances, both neglectful and in breach of his duty as a district surgeon, and merited censure and condemnation by the community and the authorities concerned;

(*h*) That in all the circumstances the plaintiff had been guilty of serious and despicable misconduct in his profession, which merited the censure of the community and all right-minded people.

The appellant claimed £5,000 as and for damages suffered by him 'to his good name and reputation in his personal and in his professional capacity'.

The respondent admitted that the meeting was held as alleged, but denied that he had spoken the words ascribed to him; alternatively, he denied the innuendo placed on them in the declaration; and as a second alternative he pleaded that, if he had published those words and if they bore the meaning assigned to them in the declaration, 'they were published by him on a privileged occasion in the circumstances hereunder set out;

'(*a*) Defendant has at all relevant times been a farmer in the Zwartberg area of Mount Currie District, and a member of the Zwartberg Farmers' Association.

'(*b*) The said Association is a voluntary association of farmers in the said area, whose objects, *inter alia*, are (i) to serve as a mouthpiece of local farmers and to act on their behalf; (ii) to deal with, further and protect the interests of the farming community; and (iii) to do all things which in the opinion of a meeting of members are in the interests of the members of the farming community.

* Judgment (1957 : S. Afr. Med. J., 31, 571 (8 June).

'(*c*) In terms of the constitution of the said Association, all matters affecting the said Association and its interests shall be dealt with at the general meeting of the said Association.

'(*d*) On 27 October 1955 the general meeting of the said Association was held at Zwartberg, and was attended by defendant and the persons mentioned in the declaration, all of whom were members of the said Association.

'(*e*) The professional conduct of the district surgeon for the said area was a matter affecting the interests of the farming community of the said area, in particular the members of the said Association.

'(*f*) The said words published by defendant related to the professional conduct of the district surgeon for the said area.

'(*g*) The said words were published by defendant at the said general meeting without malice and in the *bona fide* belief that they were true.

'(*h*) It was the duty of defendant to publish the said words to the members present at the said general meeting, and the said members had a lawful interest in having the said words published to them. Alternatively, the said words were published by defendant in furtherance of his interest as a member of the said Association, and the other members at the said general meeting had a lawful interest in having the said words published to them.'

I shall deal at a later stage with the occurrences that led up to the respondent's report to the farmers' meeting and with the evidence relating to the words in which he made his report. *Sampson, A. J.* did so in the early part of his judgment, and then said:

'It is clear that the statement about plaintiff made to the general meeting of the Association on 27 October 1955 was a highly misleading one which conveyed implications altogether untrue, implications which defendant has said he intended to convey. These implications were of a defamatory nature and there has been no attempt on the part of the defendant to show that they were not defamatory. The only serious defence to the resultant claim for damages is that of qualified privilege; in other words it is contended that the occasion of defendant's statement was a privileged occasion and that defendant made the statement without malice and in the *bona fide* belief that what was said was true.'

The learned Judge, holding on the evidence that the occasion was privileged and that the statement had not been shown to have been made maliciously or without a *bona fide* belief in its truth, found the defence of privilege established and dismissed the plaintiff's claim with costs.

THE ISSUE OF PRIVILEGE

On the question whether the occasion was privileged, I am prepared to assume the correctness of the learned Judge's finding that it was. I shall approach the case on the assumed basis that the evidence established the facts alleged in paragraphs (*a*) to (*e*) which I have quoted above from the portion of the plea setting up the privilege, and that these facts render applicable the principle of qualified privilege which has been aptly formulated in the following words:

'Where the person publishing the defamatory matter is under a legal, moral or social duty to do so or has a legitimate interest in so doing, and the person to whom it is published has a similar duty or interest to receive it, then the occasion of the publication would be privileged.'

The words are those used by *Innes C. J.* in *Ehmke vs. Grunewald* (1921 A.D. 575 at p. 581); they were quoted by *Watermeyer A. J. A.* in the course of his instructive discussion of this branch of the law of defamation in *De Waal v. Ziervogel* (1938 A.D. 112 at p. 121).

Approaching the matter, then, on the basis that the occasion was privileged, I have to consider whether, on the evidence, the appellant, as plaintiff, succeeded in establishing not only that the respondent uttered words that were defamatory of him, but also that the respondent was in fact actuated by the *animus injuriandi* which would have been presumed from the publication of defamatory words if there had been no privilege. For this purpose I shall shortly outline the facts that are relevant on these issues.

The respondent, a farmer in the Mount Currie District, testified that at about 10.20 on the night of 13 October he received a telephone message as a result of which he went immediately to the farm of a neighbour, a Mr. Cullen. There he and other neighbours, including one Stubbs, found Cullen's dead body in a motor car; he had committed suicide by the inhalation of exhaust gas.

RESPONDENT'S EVIDENCE

I now quote from the respondent's evidence, given while he was being examined in chief by his Counsel, *Mr. O'Hagan*:

'You went to Mr. Cullen's house after seeing the body in the car? . . . Correct.

And you made certain telephone calls from that house? . . . We did.

By about what time did you get back to the house? . . . About quarter to eleven.

Just tell his Lordship what calls you made? . . . We endeavoured first of all to contact our local police camp in Banchory. We had no success, so we asked our exchange, which is then Cedarville at that hour, to please try and contact Zwartberg, which is our next nearest police camp, without success. Zwartberg, we had no success there. In desperation we tried Cedarville, which is further still, and all we could contact there was a Native constable, so he was of no assistance to us. So we got through to Kokstad; it is a matter of 48 miles from the scene of the tragedy.

Now when you got through to Kokstad approximately what time was it? . . . It must have been approximately 11 o'clock.

Who did the speaking? . . . Mr. Stubbs.

Did he speak to the police at Kokstad? . . . He did.

Could you hear the conversation? . . . I could, because this phone had an extra ear-piece.

Now will you tell His Lordship what happened and what the conversation was between Mr. Stubbs and the Kokstad police? . . . My Lord, we reported this matter to the police or whoever was in charge of the charge office at Kokstad. We told him exactly what had happened, what we had found. His immediate words to Mr. Stubbs were, on no account was he to touch the body. We said, we fully realize that, would he please make arrangements for someone to come out to the scene of the tragedy? By someone we meant the police, and at the same time would he kindly contact the District Surgeon and notify him of the tragedy?

Yes, and what was the reply of the police? . . . At the same time he said he would do so and at the same time we asked him and said it is very late, the exchanges are all difficult to get through; after a certain hour we have the privilege of night service and we do not know what happened to the operators. We explained a few outlines of the circumstances and asked him to please do us a favour and keep in operation because we were expecting a return call from the charge office, Kokstad, telling us exactly what they were doing in the matter.

You were expecting the police to ring you back? . . . Definitely.

Had he said he would phone back? . . . He promised he would.

Now, Mr. Whittle, after this telephone call what did you and Mr. Stubbs do? . . . My Lord, we waited until approximately 1 a.m. and there had been no notification, no phone calls from Kokstad or anyone else.

Yes? . . . We then thought it time to put a reverse call back to Kokstad and ask them what they were doing.

Who put that call through? . . . Mr. Stubbs put a call through and I listened in the extra ear-piece.

Continue: . . . Mr. Stubbs asked the same gentleman—it sounded like the same gentleman. I cannot swear, but the voice sounded the same—asked him, what have you fellows done in the matter. He said, we are still attempting to get someone to go out, which rather shocked us. It was from 11 to 1 o'clock, a matter of 48 miles and they were still looking for somebody to come out.

By Sampson, A. J.:

From Kokstad? . . . To come out to the scene of the tragedy.

The police? . . . The police, yes. Mr. Stubbs said I presume the District Surgeon is on his way, and the reply we received was that the District Surgeon would not be out until tomorrow morning.

Mr. Stubbs then said, I presume the District Surgeon is coming? . . . Yes, Mr. Stubbs asked that question twice.

Then the voice said? . . . The District Surgeon will not be coming out until tomorrow morning.

Will not be out until 12 tomorrow morning? . . . Not until tomorrow morning.

What did Mr. Stubbs say? . . . He said, what is that? He repeated, you see the District Surgeon will not be out until tomorrow morning.

Did the voice then say anything? . . . Nothing. I then said to Mr. Stubbs, let us put down and wake the Commandant.

Did he say the District Surgeon refused to come out? . . . He said the District Surgeon will not be out until tomorrow morning. We were both shocked or surprised.

Then you hung up? . . . No, we then got in touch with Captain McGregor.

By Mr. O'Hagan:

And he is? . . . He is the District Commandant at Kokstad. The District Commandant's first words to us were: 'Have you been in touch with the office concerning this matter?' I think he was rather annoyed and we said, yes, we have been in touch with the office. He said, in that case I will see that somebody comes out immediately.

Yes? . . . And at approximately quarter to four a.m., first of all the next of kin arrived and then at 4 o'clock the District Commandant and Sergeant Brand arrived, that is 4 a.m. from 11 to 4 o'clock.

In the meantime had the body of Mr. Cullen been touched or moved? . . . We were instructed not to touch anything. We left it in the car on the banks of the river with a Native boy in charge.

When the District Commandant arrived what did he do? . . . We discussed the matter—it is some considerable way from the house—and then proceeded to the scene of the tragedy.

And ultimately you and Mr. Stubbs were given authority to take the body to Kokstad? . . . Yes. by the District Commandant.

And I think you took the body in early the next morning? . . . We took the body to the District Surgeon, Kokstad. We got in approximately 6 o'clock in our pyjamas and overcoats.

Now after this action you reported the matter to the Annual General Meeting of the Association? . . . I did.'

MESSAGE RECEIVED BY RESPONDENT AT CHARGE OFFICE

The appellant's evidence—which on this point was not contradicted—nor was it questioned by the defence—was that, although he had been at home during the evening and the night of 13 October, he had received no message that night, and Cullen's suicide, with the intimation that he should do a *post mortem*, had only been reported to him at about half past eight on the morning of the 14th.

The wording of the message which the respondent and Stubbs received from the Kokstad charge office in the night of 13 October is given nowhere else in the record than in the respondent's evidence. No police witness was called at the trial. Stubbs was called by the defence, but he did not give the words of the message.

The respondent was afforded several opportunities to state what the message was, and he repeated it in the same words every time: Mr. Stubbs said, I presume the District Surgeon is on his way, and the reply we received was that the District Surgeon would not be out until tomorrow morning.

What the respondent is alleged in the declaration to have said at the meeting is that 'the District Surgeon was telephoned at 10.30 p.m. and refused to appear at that hour, although the circumstances were explained'.

It is clear from the respondent's evidence that the only information on which he based his report to the meeting was the message received from the man on duty at the Kokstad police station at 1 o'clock in the night of the 13th.

That message did not say that a telephonic communication had reached the appellant at 10.30 p.m. or at all. For all the message said, the appellant might not have been at home and the police might not have been able to get into direct contact with him or might for some other reason in fact not have done so. His home, indeed, was not at Kokstad, but at Franklin. The appellant's evidence, not disputed, was to the effect that his practice serves an area with a radius of 40 miles, containing about six or seven hundred Europeans and about seventeen thousand Natives, and that he pays regular visits, on certain days of the week, to various clinics situate in different directions at considerable distances from his Franklin headquarters. There certainly was nothing in the police message to suggest that the appellant had *refused* to come. Even if he had received the message, he might have been unable to come immediately. He might well have been held up by a difficult confinement or some other critical case.

It was in this allegation of the *refusal* to come, which was not justified by the police message, that the sting of the alleged defamatory statement lay.

WORDS USED BY RESPONDENT AT THE MEETING

It is therefore necessary to consider whether the evidence proves that the words used by the respondent at the meeting were those complained of in the declaration or words to a like effect.

The words in the declaration are taken *verbatim* from a letter dated 31 October 1955, sent to the appellant by the magistrate of Kokstad, who had copied them from a letter dated 29 October written by the secretary of the Zwartberg Farmers' Association to the magistrate. The secretary had written this letter in compliance with a resolution adopted at the Association's meeting of 27 October as a direct result of the report made to it by the respondent. Clearly, therefore, it represented the secretary's impression of what the respondent had said. In the minutes of the meeting he had recorded the relevant portion of the respondent's report in the words:

'They were informed that they (the Kokstad police) were unable to contact any policeman to send out and the medical officer at Franklin had refused to go out.' The Secretary, one Barnes, was called as a witness for the respondent. He said that the respondent had said at the meeting that 'the District Surgeon refused to go out that night or words to that effect'. Nowhere in his evidence did he suggest that his written version, in the minutes and in his letter, was based on any information other than the respondent's report to the meeting or did not truly reflect the tenor of that report. The only other witness, apart from the respondent, who testified about the words used at the meeting was a witness for the appellant, one Mortlock. According to him, the respondent had said that when they had got the police at Kokstad the first time they had reported the matter (of the suicide) requesting them to get through to Franklin:

'Mr. Whittle, the defendant, instructed him' (the policeman) 'to get into touch with the District Surgeon acquainting him with the facts. They wanted the District Surgeon out. This poor man was dead in the car. Several hours went by; once more he was able to get in touch with Kokstad; the policeman said he had already been through to Dr. Naudé, explained the circumstances to him, but Dr. Naudé refused to come out.'

He admitted that he could not remember the precise words. When *Mr. O'Hagan* came to cross-examine him, however, the only point on which he questioned the accuracy of the witness's recollection of what the respondent had said was whether he had not said that it was Stubbs who had spoken over the telephone while it was the respondent who had listened on the extra earpiece; the witness had put it the other way about. It was not suggested to the witness that he had been wrong in saying that the respondent had reported the policeman as having said that Dr. Naudé 'refused to come out'.

At an earlier stage, while the appellant—who was the first to give evidence in the case—was in the witness-box, *Mr. O'Hagan*, in the course of his cross-examination of the appellant said:

'You see Mr. Whittle's case is going to be this. I must put it to you that Mr. Whittle and Mr. Stubbs, who were together when Mr. Cullen's body was found, who together phoned the police and asked for the police and for the District Surgeon, they received this report from the police that the District Surgeon would not come out and Mr. Whittle conceived it to be his duty to report this report of the police to the Association; and Mr. Whittle will say he has nothing at all against you but he felt it his duty to put it to the Association so that they could take whatever action was required in the matter, whatever investigation was necessary.'

RESPONDENT'S EVIDENCE (CONTIN.)

I now quote from the examination-in-chief of the respondent:

'*By Mr. O'Hagan:*

Mr. Whittle . . . when you overheard the Kokstad police make this remark to Mr. Stubbs that the District Surgeon would not be out until tomorrow what did that convey to you, what did you understand? . . . I understood possibly by the remark that he refused to go out.

Is that what you understood? . . . What could I conclude? He may have been ill. They said the District Surgeon would not be out.

When you made that statement to the meeting what meaning did the persons present at the meeting assign to that statement? . . . My Lord I am not a thought-reader but all I can suggest is that possibly they may have had different opinions but they possibly

all concluded that the District Surgeon refused to go out, but I did not take part in any further discussion over this matter.'

The learned judge then wished to know from Mr. O'Hagan whether he was correct in his impression that the respondent had said to the meeting that he was told by the Kokstad police that the District Surgeon had refused to go out. *Mr. O'Hagan* replied in the affirmative, and added:

'In case there is any misunderstanding I want to make it perfectly clear that I am not suggesting in this case that there was a non-defamatory remark made. We are not setting up the case that we made a perfectly innocent remark which was misinterpreted by the meeting. We conveyed a report, whatever particular words we used, that the District Surgeon refused to go. That is, I am not making any other sort of suggestion in the case.'

The respondent, who was in the witness-box at the time, continued his evidence without making any comment on his counsel's statement to the Court.

In cross-examination the respondent said: 'I said that the police had informed me that the Doctor had refused to come out.'

I have already quoted the portion of his evidence in which he related what the police had told him. It was merely: 'The District Surgeon will not be out until tomorrow morning.'

I have referred at some length to the evidence of these two aspects of the case—what the policeman had actually said and what the respondent had reported to the meeting as having been said by the police—because at several stages in his evidence the respondent took up the attitude that he had merely repeated to the meeting what the police had said.

I quote again from his examination-in-chief:

'*By Mr. O'Hagan:*

Yes? . . . I then explained to the Association that I had gleaned my information from the police concerning the District Surgeon, that we have requested that this officer, whoever he may be, in the office to notify the District Surgeon and tell him of this tragedy and that the District Surgeon would be of value. We got the reply that the District Surgeon would not be out until the following morning. I then sat down and it was for my Association to discuss the matter, which they did.

When you heard the police say over the telephone that the District Surgeon would not be out until tomorrow, when you heard that statement made, did you people believe that the District Surgeon had refused to come out?

Mr. Cloete: He has not said anything about refusal.

Sampson, A. J.: What did you say, Mr. Cloete?

Mr. Cloete: There is no question being said about the District Surgeon having refused to go out and I do not think my learned friend . . .

Sampson, A. J.: What the witness said is that he told the meeting that he was informed by the police that the District Surgeon would not be out until the following morning.

Mr. Cloete: Yes, My Lord.

Mr. O'Hagan:

Well, I will put it this way, what did you understand by the statement of the police? . . . I understood that the District Surgeon naturally had refused; what other construction could I construe from the statement?

What I want to ask you is this, did you believe in your own mind that that was in fact the case when you heard the police say this? . . . If we cannot believe the police.

By Sampson A. J.: Just a minute. From your evidence I understand, put it that way, that you told the meeting what the police had actually said to you? . . . That the District Surgeon would not be out until the morning.

Are you quite sure that you did not say he refused to go? . . . The way I put it, but that he would not be out until the morning.

But you heard a number of witnesses, at least two witnesses, who gave the statement that you said the District Surgeon had refused to go, that was the report you got? . . . If they wish to interpret it that way I cannot help it.

Now is it possible that after this statement of yours other people in speaking about what happened had drawn the conclusion that you drew, which I think was the natural conclusion, that they did? . . . Yes.

That others made that remark? . . . My Lord, this matter was open for discussion, I did not say anything further, I sat down. It was for the meeting and my Association to take the necessary steps.'

The tenour of this part of the respondent's evidence was clearly that all he had reported to the meeting was that the police had said that the District Surgeon would not be out until the morning, and that if others understood from this that the doctor had refused to come, that was their own interpretation.

I need hardly point out that this is in conflict not only with the evidence of Mortlock and Barnes, which was not questioned by the respondent's counsel, but also with other quotations which I have already given from the respondent's own evidence and with the statements made on his behalf by his counsel.

In cross-examination he took another line. He made the direct admission I have already quoted: 'I said that the police had informed me that the Doctor had refused to come out', but when it was put to him that this was a serious thing for a District Surgeon to do, he gave answers which implied that it was what the police had told him: 'I received this message from an authentic source . . . I did not concoct it, they told me so.'

The learned Judge found 'that the terms of the report in the minute reflect the effect of the actual words as this effect was voiced in the discussion that followed'.

According to the minute the respondent had reported: 'They were informed that . . . the medical officer at Franklin had refused to go out.'

ALLEGED REFUSAL TO ATTEND NOT VERIFIED

In view of the evidence I have quoted and discussed, I do not see how this finding can be queried, and, indeed, *Mr. O'Hagan*, in his argument before us, conceded its substantial correctness.

Nor does the evidence leave any doubt in my mind that the allegation of a *refusal* on the appellant's part to come was not contained in the police message, but was the respondent's own gloss on it. And, as I have said before, the entire sting of the imputation lies in this allegation.

In making this addition to the police message the respondent, to my mind, showed a reckless indifference both to the question whether the imputation contained in it was true or false and to the harm it might do the doctor.

There is no suggestion in the respondent's evidence that he had misunderstood the policeman and had mistakenly formed in his own mind the impression that the policeman had said that the doctor had *refused* to come. When the case was heard in February 1957, 16 months after the occurrence, he could still repeat the words the policeman had used. Those words, as he repeated them, did not contain the imputation that the doctor had refused. On his own evidence that imputation was a construction he himself had put on the policeman's words.

It was a construction he could very easily have verified. A fortnight elapsed between the telephone conversation and the meeting to which he reported. In that interval he could have telephoned the police to get further particulars from them as to why the doctor had not come out, or he could have telephoned the appellant himself to get an explanation from him. The respondent himself mentioned in his evidence the possibility that the doctor might have been ill. Yet, while there might have been many innocent reasons why the doctor was not coming till the next day, the respondent chose one that cast a serious slur on the doctor's professional conduct and put that to the meeting as the only reason, and, what is more, as the reason that had been given by the police.

The respondent's counsel is recorded as having said:

'He felt it his duty to put it to the Association so that they could take whatever action was required in the matter, whatever investigation was necessary.'

It would have been a very simple matter for him to make the investigation himself before putting the worst construction on the message; and, at most, the duty, if he felt any, would have been to repeat the message as it had been given to him, not to add a defamatory imputation which it had not contained.

He was under no illusion as to the gravity of the imputation; indeed, his whole object in putting it to the meeting was to have a complaint against the doctor made to the magistrate, and he admits that the resolution to do so—which was acted upon—was unanimously passed by the meeting while he was present as a member.

QUESTION OF ANIMUS

Can it be said, on these facts, that he acted without *animus injuriandi* and was therefore protected by the privilege of the occasion?

In *Findlay v. Knight*, 1935 A.D. 58, the Appeal Court had to consider a case in which matter defamatory of one party to a lawsuit had been contained in a plea drawn and filed by the attorney of the other party. *Wessels C. J.*, after referring to decisions of our Courts, at pp. 71 and 72, quoted passages from *Voet* and *Van Leeuwen* which are illustrative of the limits of qualified privilege. He mentioned *Voet* 47.10.20, where the writer says that if charges are made by a litigant to impugn the credibility of a witness who testifies against him, he should not be considered to be doing so *injuriandi animo* '*si modo verisimiles aliquas opprobrii facti causas producere possit*'—provided he can produce a reasonable foundation for the defamatory charges. Having considered the authorities, the learned *Chief Justice* said:

'The pleader who does not care whether his libellous charge is true or false, and who knows or ought to know that no evidence will be produced to support his charge is in no better position' (than one who knows the charge to be false). 'He is manifestly abusing the process of the Court and using the freedom of a pleader for some ulterior or improper motive which it is unnecessary for the Court to formulate, because if he then publishes a libel recklessly without caring whether it is true or false he must be presumed to act *animo injuriandi*.'

Curlewis J. A. (at p. 73) and *de Villiers J. A.* (at p. 76), while concurring in the result, made it clear that they wished to leave open the question—on which they felt some doubt—whether an attorney would be liable 'in cases where he is instructed, or knows, or believes, or assumes, that there will be some evidence to support the defamatory allegation, but at the same time he makes the allegation recklessly, in the sense that he does not care whether it is true or false'.

In the case before them the attorney had known, when he filed the plea, that there would be no evidence to support the defamatory allegation.

In the present case the respondent, when he reported the police as having said that the appellant had refused to come, knew that the message from the police had not contained that allegation. I therefore need not consider the effect of the reservation made by *Curlewis J. A.* and *de Villiers J. A.*

In *Monckton v. British South Africa Co.*, 1920 A.D. 324, *Innes C. J.* said at p. 332:

'In the majority of cases a defendant who exceeds his privilege is actuated by what in English law is called express malice; but *animus injuriandi* may be established not only by proving actual ill-will towards the plaintiff, but by showing that the defendant was actuated by an indirect or improper motive or that he stated what he did not know to be true, reckless whether it was true or false.'

The decision in *Findlay v. Knight* was considered and followed in *Gluckman v. Schneider*, 1936 A.D. 151, in which a reference was also made to *Monckten's* case—*vide* judgment of *Stratford J. A.* at pp. 158, 160, 162. There (in *Gluckman's* case) the alleged defamation had occurred in the course of the cross-examination of a Crown witness by the attorney defending the accused in a criminal case. The attorney had some information about alleged thefts having been committed by the witness, and he emphatically charged him with them. The witness sued the attorney for defamation, and the judgment given against the attorney was upheld by the Appeal Court, which considered that the information he had received was of so unreliable a nature that he should not have acted upon it, in the definite way he did, without verifying it (see pp. 162-164). The head-note says:

'*Held*, on appeal, that the defendant had made the accusations recklessly without caring whether they were true or false and without reasonable grounds for believing them to be true and that although the occasion was privileged *animus injuriandi* had correctly been inferred by the court below.'

The words are those of the writer of the head-note, but I think they correctly summarize the effect of the judgment.

There may be cases in which a man makes a defamatory statement without reasonable grounds for belief in its truth, but does so under circumstances which negative the inference of *animus injuriandi* or—to use the term which *Schreiner J. A.* in *Basner v. Trigger*, 1946 A.D. 83, at pp. 95-96, considered more apt in this connection—what English law calls 'malice' in the sense of any improper or indirect motive. Such a case was the one I have just mentioned. There the defamatory statement occurred in the course of an argument addressed by the defendant—appellant in the Appeal Court—to a tribunal before which he was appearing in the interest of certain groups of people whose case he was presenting. The statement was one of several deductions he purported to draw from a written report which the plaintiff had made. The tribunal had the report itself before it. I quote from the judgment of the Court delivered by *Schreiner J. A.* at p. 107:

'It is, unfortunately, true that arguments are sometimes, from lack of experience, presented in the form of expressions of the arguer's belief or opinion but essentially he is throughout putting forward submissions as to the weight of the evidence and the inferences to be drawn therefrom. Considerable latitude must be allowed to the party or his representative who is thus presenting his case and trying to persuade the tribunal to his view. Malice is not to be attributed to him merely because he does not think his submissions well founded or because they are pitched too high for reasonable acceptance.'

The distinction between *Basner's* case and the one before us is obvious. The present respondent was not arguing a case and submitting his interpretation of or deductions from facts known to his hearers, who could therefore judge for themselves whether his statements were justified or not; he was purporting to state a fact, i.e. what the police had told him; he was conveying to them information of which they had only his version to go by.

The impression I get is that the respondent worked himself up into such a state of annoyance when he and Stubbs failed to get the immediate responses he apparently expected from the telephone service, the police and the District Surgeon that he did not even try to form a calm and dispassionate judgment but decided to have them all taught a lesson. It seems to me to have been that frame of mind, and not a sense of duty, which led him to put on the message he had received a sinister construction not warranted by its actual content, and then to pass his construction on to the farmers' meeting as if it was the actual wording of the message. That was an abuse of whatever privilege the occasion might have afforded him.

APPEAL SUCCEEDS

It follows that the appeal should succeed, and the only remaining question is the amount of damages that should be awarded. On this point the trial Judge said:

'If, however, I am wrong in finding that the occasion of its publication was privileged, I would after consideration of all the circumstances: the absence of apology in public, the failure of plaintiff to respond to the offer contained in the letter of 15 December 1955, the fact that Mr. Dorning says that when he stated to the meeting of the Association that the report was false, practically everyone was aware of the fact, and the consideration that the publication did not have a very wide ambit of interest and was probably quickly corrected so far as the Franklin and Zwartberg area was concerned, allow in damages the sum of £800.'

I have dealt with the case on the basis that the learned Judge was right in holding the occasion to have been privileged but was wrong in not finding that the privilege had been abused. For the purpose of determining the *quantum* of damages, however, I see no reason for drawing a distinction in this case between a liability arising from absence of privilege and one arising from abuse of privilege.

The letter of 15 December 1955 referred to by the learned Judge was one in which the Association, through its attorneys, offered to send out a circular to its members correcting the report that the District Surgeon was ever telephoned or knew of the late Mr. Cullen's death on the night in question. The amount assessed by the learned Judge which was 'in the nature of things necessarily a matter of estimate', should not be disturbed unless we consider that, as an estimate, it is wrong (*vide S.A. Railways v. New Silverton Estates Ltd.*, 1946 A.D. 830 at p. 838). I cannot say that, taking all factors into account, it is not a fair estimate, and I therefore accept it.

In the result the appeal is allowed with costs and the judgment of the Court *a quo* is altered to read: Judgment for the Plaintiff in the sum of £800 with costs.

Steyn J. A.
Malan J. A.
Reynolds A. J. A.
Hall A. J. A.
} concur

RESEARCH FORUM

STUDIES IN CHOLESTEROL TRANSPORT AND CATABOLISM

B. LEWIS, M.B., CH.B. (CAPE TOWN)

At a meeting of the Research Forum of the University of Cape Town held at Groote Schuur Hospital, Cape Town, on 4 December 1957, papers were read by Dr. B. Lewis and by Mr. J. A. Wilkens on behalf of the members of the C.S.I.R. Clinical Nutrition Research Unit, Department of Medicine, University of Cape Town. The following are summaries of the two papers:

At an earlier meeting[1] of this Forum (5 June 1957) data were presented showing that the reduction of serum-cholesterol concentration by sunflower-seed oil feeding was accompanied by an increase in faecal bile-acid elimination. The bile-acid increments accounted for roughly two-thirds of the cholesterol leaving the circulation. It is known that cholic acid is the principal end-product of cholesterol metabolism appearing in the bile, but its faecal derivatives remain to be characterized precisely; determinations on faeces can thus give only an approximate measure of cholesterol breakdown.

We have therefore carried out somewhat similar experiments on 5 subjects with bile fistulae, who were fed with hydrogenated coconut fat (HCO), sunflower-seed oil (SSO) or carbohydrate in addition to their basal low-fat diet. One subject received cotton-seed oil (CSO) emulsion intravenously. The HCO had an iodine value of 6, the SSO 135, and the CSO 115. The emulsifying agent was shown to have no effect on the serum-cholesterol concentration. Bile acids were determined in bile by a highly selective column chromatographic procedure evolved by Dr. S. Mirvish of the Department of Physiology, Witwatersrand University. The elevation of serum-cholesterol concentration due to HCO was not accompanied by notable changes in bile acid or biliary cholesterol output. On the other hand, reduction of the serum-cholesterol concentration by SSO or CSO was preceded and accompanied by a striking increase in cholic acid excretion from a mean basal figure of 5·6 g./24 hours to a mean of 16·3 g. in 24 hours during oil feeding, and an inconsistent and minor increase in deoxycholic or cheno-deoxycholic acid. Bile cholesterol showed no consistent variation. While this effect of SSO and CSO may be interpreted as indicating an increased catabolism of cholesterol, the rise in cholic acid elimination was considerably in excess of the amount needed to account for the fall in serum cholesterol; either cholesterol was mobilized from the tissues or more probably its synthesis was accelerated; for the latter hypothesis there is experimental evidence.

Because plasma cholesterol esters contain principally those fatty acids which are highly unsaturated, which the mammalian body cannot synthesize and which are present in large amounts in SSO and CSO, a technique was developed for analysis of cholesteryl-ester fatty acids (CEFA) based on chromatography on silica gel, alkali isomerization and a micro-bromine addition procedure. It was readily established that the CEFA composition of the serum reflected the nature of the dietary oil, iodine values falling to 115 during HCO feeding and rising to 208 when SSO was administered. Linoleic and arachidonic acids showed the greatest change.

A blind survey was carried out on normal Bantu and White subjects, and on age-matched ambulant patients with coronary heart disease. The study is still in progress, but available data showed a trend towards increasing unsaturation in the order coronary patients, normal Whites, Bantu; the mean iodine values (with standard deviation) being 136 (35·6), 162 (34·5), 179 (26·5).

CEFA obtained from atheromatous coronary arteries and analysed by the same procedure had a mean iodine value of 64.

It may be speculated that esters of cholesterol with highly unsaturated fatty acids are more 'soluble' in plasma than less unsaturated ones, and that the former are more active metabolically and hence more rapidly catabolized.

[1] Report (1957): S. Afr. Med. J., 31, 594.

THE EFFECT OF DIETARY LIPID FRACTIONS ON THE SERUM CHOLESTEROL OF THE RAT

J. A. WILKENS, B.SC.HONS.

1. In a series of experiments the suitability of the rat was tested as an experimental animal for the investigation of dietary effects on the serum-cholesterol level. Rats were fed on a diet based on skim-milk powder to which 15% to 20% of various proportions of coconut fat and sunflower-seed oil and their fractions were added.

2. It was found that dietary coconut fat raised the serum cholesterol to approximately 30% above the fat-free isocaloric diet. On replacing increasing proportions of coconut fat by sunflower-seed oil the raising effect of the former was progressively neutralized. There were no significant differential weight changes between groups receiving equal amounts of different fats, demonstrating uniform absorption of the diet.

Under the conditions of the experiments it was not always possible to maintain differential effects on the serum-cholesterol level beyond 30 days.

3. No cholesterol-lowering substances were detected either in husks or in the non-oily part of the kernel comprising the whole sunflower seed.

4. In a further experiment the sunflower-seed oil was saponified, the non-saponified (or non-fatty) matter separated, and the resulting pure soaps converted to the ethyl esters of the fatty acids. These fractions could then be tested separately and in various combinations.

It was found that a diet of pure coconut fat to which 0·4% of the unsaponifiable matter separated from sunflower-seed oil was added slightly lowered the serum cholesterol of rats, and was not significantly different in this respect from a diet of crude sunflower-seed oil fatty-acid esters from which the unsaponifiable matter had not been removed.

The purified ester diet substantially freed from unsaponifiable matter resulted in a serum cholesterol intermediate between crude sunflower-seed esters and coconut fat.

5. In a last experiment the unsaponifiable matter of coconut and sunflower-seed oils were interchanged, the fat fractions being fed as their ethyl esters. The coconut-fat ester with sunflower-seed unsaponifiables completely suppressed the cholesterol-raising property shown by crude coconut ester with its unsaponifiables after 9 days' feedings, and to the extent of 60% of the 18 days'. No significant differences in the cholesterol levels were found between groups of rats on diets of crude sunflower-seed oil esters, and pure sunflower-seed oil esters containing coconut-fat unsaponifiables, both diets producing a mean depression of 5%.

6. It is concluded that at least a part of the dietary hypocholesterolaemic effect of sunflower-seed oil on the rat may reside in the unsaponifiable (or non-fatty) matter of the oil.

NEW PREPARATIONS AND APPLIANCES: NUWE PREPARATE EN TOESTELLE

'PROTAMYL' BRAND PROMETHAZINE-AMYLOBARBITONE TABLETS

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of 'Protamyl' brand promethazine-amylobarbitone tablets, each containing 12·5 mg. of promethazine hydrochloride and 50 mg. of amylobarbitone; and supply the following information:

Preliminary clinical studies were aimed at finding the proportion of the constituents which would enable the product to be given to out-patients in a dosage which would ensure sleep at night and provide sedation in the daytime with minimal side-effects.

The full clinical effect of amylobarbitone is achieved with much reduced dosage in 'Protamyl'; this, together with the established safety of promethazine, which has many of the clinically useful properties of chlorpromazine, makes the association of particular interest in general medicine because of its value in the treatment of anxiety and tension states.

'Protamyl' is indicated mainly in the less severe mental and emotional disturbances, for use at bedtime in cases of insomnia, in obstetrical sedation, and in certain dermatological conditions particularly chronic urticaria and atopic eczema.

'Protamyl' is supplied as pale-lilac sugar-coated tablets in containers of 50 and 500.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. Marcus Fredman, B.Sc., M.B., F.R.C.S. (Eng.) has commenced practice as a Surgical Specialist at 1021, Medical Centre, Cape Town. Telephones: rooms 33466, residence 445072.

* * *

The South African Society of Occupational Health. A short course on 'the Elementary Principles of Statistics as applied to Medicine' will be given by Dr. A. M. Adelstein in the Council Chamber, Medical House, 5 Esselen Street, Johannesburg on Tuesdays 28 January and 4, 11 and 18 February 1958 at 5.15 p.m. Enquiries should be directed to Dr. B. Serebro, 131 Union Centre, 31 Pritchard Street, Johannesburg.

* * *

Mev. J. Louw, B.Sc. Huish. (Pret.), M.Sc. (Columbia), Dipl. American Dietetic Association, Regd. Dieetkundige S.A. Mediese Raad, en mev. E. E. Versluis, B.Sc. Huish. (Pret.), Dipl. American Dietetic Association (Duke), Regd. Dieetkundige S.A. Mediese Raad, maak hiermee bekend dat hulle met ingang 1 Februarie 1958 as Konsulterende Dieetkundiges te Mediese Sentrum 418, Pretoriusstraat, Pretoria, sal praktiseer. Telefoon 33444. Ure: Maandae tot Vrydae, 9 vm. tot 12.30 nm.

* * *

Ciba Limited, Basle, Switzerland, announce the formation of a South African subsidiary company, the Ciba (Pty.) Ltd., City Centre, corner of Kerk and Sauer Streets, Johannesburg. The new company will be concerned with sales and production of Ciba pharmaceutical specialities in South Africa. Sana Limited have acted as agents for Ciba for the past 10 years, and have assisted in the formation of the new company. Certain changes in the arrangements for distribution have been made and are now in operation; Ciba have sent full details to the pharmaceutical trade.

* * *

Dr. Pierre du Toit, M.B., Ch.B. (Cape Town), M.R.C.O.G. (London), M.O. & G. (Cape Town), has relinquished his private practice in Cape Town, having been appointed senior full-time gynaecologist to the Karl Bremer Hospital, Bellville, Cape, and the Medical Faculty of the University of Stellenbosch.

Dr. Pierre du Toit, M.B., Ch.B. (Kaapstad), M.R.C.O.G. (Londen), M.O. & G. (Kaapstad) is aangestel as senior voltydse ginekoloog te Karl Bremer-Hospitaal, Bellville, Kaap, en die Mediese Fakulteit van die Universiteit Stellenbosch en staak dus nou privaatpraktyk.

* * *

Research Forum. The following meetings of the Research Forum, University of Cape Town, will be held at Groote Schuur Hospital, Cape Town, in the A-Floor Lecture Theatre: On Wednesday 29 January at 12 noon, when the subject will be 'The regulation of the serum-cholesterol level in normal active men: long-term effect of certain saturated and unsaturated fats and of other environmental variables', and the speakers will be Dr. H. Gordon and Prof. J. F. Brock. On Wednesday 5 February at 12 noon, when the subject will be 'potassium and sodium balance in relation to paralysis', and the speakers will be Prof. L. Eales, Prof. G. C. Linder and Dr. B. Sakinofsky.

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 25 December 1957-2 January 1958.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	7	2	–	–	9
Cape Province ..	2	3	1	–	6
Orange Free State ..	1	–	–	–	1
Natal	–	–	–	–	–
Totals	10	5	1	–	16

Plague, Smallpox, Typhus Fever: Nil.

* * *

By die Jaardag van die Karl Bremer-Hospitaal, Bellville, Kaap gehou op 4 Oktober 1957, is 'n toekenning vir die mees oorspronklike wetenskaplike uitstalling gemaak aan dr. J. N. de Klerk vir 'n rolprent met die titel ‚Uretra-plastiek'; en 'n toekenning vir die mees oorspronklike navorsingswerk is gemaak aan dr. J. Roux vir sy bydrae oor ‚Die Etiologie van Toksemie van Swangerskap'.

At the Year Day at the Karl Bremer Hospital, Bellville, Cape, held on 4 October 1957 the award for the most original scientific exhibit was made to Dr. J. N. de Klerk for a film entitled Urethroplasty; and the award for the most original research work was made to Dr. J. Roux for his contribution on 'Aetiology of Toxaemia of Pregnancy'.

* * *

The Commonwealth Medical Advisory Bureau, established in 1948 by the British Medical Association with a view to welcoming and providing a personal advisory service for practitioners visiting the United Kingdom from the Dominions and Colonies has published a 30-page brochure (C.M.A.B. 11/11 Dec. 1957) entitled 'Summary of Regulations for Postgraduate Diplomas and of Courses of Instruction in Postgraduate Medicine'. Besides the actual Summary of Regulations etc. the brochure contains information of all kinds which may be useful to medical graduates who contemplate undertaking postgraduate work or examinations in Great Britain or Ireland. Copies may be obtained from Dr. R. A. Pallister, *O.B.E.*, M.D., M.R.C.P., D.T.M. & H., Medical Director, Commonwealth Medical Advisory Bureau, B.M.A. House, Tavistock Square, London, W.C. 1, to whom all communications should be sent.

* * *

International Congress on Pneumoconiosis. The Council for Scientific and Industrial Research acting on behalf of the Government is convening an international conference on pneumoconiosis to be held in Johannesburg in 1959. The conference will be the first of its kind to bring together medical, engineering, chemical, physical and radiological experts to discuss all aspects of the pneumoconiosis problem. Several foreign governments and organizations such as WHO and the ILO are expected to send delegates. Prof. S. F. Oosthuizen, Chairman of the South African

Medical and Dental Council will be President of the conference and the Secretary-General will be Dr. A. J. Orenstein, Director of the CSIR Pneumoconiosis Research Unit in Johannesburg.

The Conference will have two themes, viz. (1) to review what has already been learned but not published, and (2) to discuss ways and means of solving outstanding problems.

South Africa as a leading mining country has for the past 40 years played a prominent part in pneumoconiosis research and prevention. The CISR Pneumoconiosis Research Unit, which is maintained by funds provided equally by the Government and the Transvaal and Orange Free State Chamber of Mines, is as comcomprehensive in its scope as any in the world. It is, therefore, particularly fitting that South African scientists should play a leading part in the coming conference, and that this conference should be held in the Union. Medical and other scientific experts in the subject have been invited to the Conference from the UK, the USA, Belgium, Germany, France, Switzerland and Northern Rhodesia.

REVIEWS OF BOOKS : BOEKRESENSIES

BLOOD DISEASES

Disorders of the Blood: Diagnosis: Pathology: Treatment: Technique. Eighth Edition. By Sir Lionel E. H. Whitby, C.V.O., M.C., M.A., M.D. (Cantab.), F.R.C.P. (Lond.), D.P.H. and C. J. C. Britton, M.D. (New Zealand), D.P.H. Pp. viii + 878. 124 Figures. 75s. net. London: J. & A. Churchill Ltd. 1957.

Contents: I. The Origin, Development, Functions and Fate of the Cells of the Blood. II. Abnormal Haemopoiesis and Abnormal Cells found in the Circulation. III. The Principles and Practice of Haematological Diagnosis. I. Red Cells. IV. The Principles and Practice of Haematological Diagnosis. II. Leucocytes and Blood Platelets. V. The Principles and Practice of Haematological Diagnosis. III. Physical and Chemical Properties of the Blood Cells and Plasma. VI. The Biochemistry and Cytochemistry of Haemopoiesis. VII. The Causes of Anaemia. VIII. The Therapeutics of Anaemia. IX. Idiopathic Hypochromic Anaemia. The Plummer-Vinson Syndrome. Chlorosis. X. Pernicious Anaemia and Nutritional Megaloblastic Anaemias. XI. Anaemias due to Disease of the Alimentary Tract and its Associated Organs. XII. Miscellaneous Dyshaemopoietic Anaemias. Radium and X-rays. Scurvy. Thyroid Disease. XIII. Haematological Aspects of Pregnancy. XIV. The Haemolytic Anaemias. XV. The Purpuric and Haemorrhagic Diseases. XVI. Anaemias in Infancy and Childhood. XVII. Diseases due to Aplasia or Hypoplasia of the Bone Marrow. Aplastic Anaemia. Agranulocytic Angina. XVIII. Polycythaemia, Erythraemia and Erythrocytosis. XIX. The Leukaemias (Leucoses). XX. Miscellaneous Disorders associated with Splenomegaly. Splenic Anaemia, Hodgkin's Disease. Diseases of Lipoid Metabolism. XXI. Infection and Infectious Diseases. XXII. Blood Groups. Haemagglutination. Blood Transfusion and Immunohaematology. XXIII. Miscellaneous Conditions. Allergy. Nephritis. Coronary Thrombosis. Diabetes. Cancer. XXIV. Disorders involving the Blood Pigments Enterogenous Cyanosis. Porphyria and Porphyrinuria. XXV. Technique. Subject Index. Index of Authors.

This is an old and trusted friend. Ever since the first edition appeared in 1935 it has been a byword amongst students and practitioners. 'Whitby and Britton' has been quoted more often than most books on haematology especially in Britain and the Commonwealth. This new (8th) edition has acquired a degree of 'middle-age spread' and is no longer able to boast of the sylph-like figure of its youth; nor is it as cheap. Nevertheless it still remains one of the best books in this field which is available today.

One can of course always find details to criticize but one has to look a little more closely in this edition! Factor 5 is not reduced by the use of the anticoagulant drugs commonly prescribed nor is factor 7 believed to be required for thromboplastin generation. The coagulation time is not almost normal between attacks of bleeding in haemophilia; no one has been able to demonstrate any difference between the coagulation functions in the usual case of haemophilia when these are measured during a bleeding phase and during a quiescent phase. 'Gaisboks disease' appears to be a diagnosis which might well be discarded while the advice to eschew liver, kidney, and meat protein in this condition can safely be ignored in the absence of uraemia. The needle which is immersed in alcohol between successive finger pricks also belongs to an age which preceded the knowledge of icterogenic viruses.

But the chief criticism I would make is that while the authors review the evidence for and against many concepts in haematology they do not indicate often enough which one they most favour. Their difficulty is plain to see for one needs to be an expert in each of these fields to give a truly authoritative opinion. This is the strength of the text book which has multiple authors. One wonders, especially since the recent untimely death of Sir Lionel Whitby, whether the time has not now come to enlarge the panel of contributors.

C.M.

DERMATOLOGIC FORMULARY

Dermatologic Formulary. Second Edition. By Frances Pascher, M.D. Pp. xii + 172. $4·00. New York: Paul B. Hoeber, Inc. 1957.

Contents: Preface. Introduction. Acknowledgments. Prefatory Notes and Abbreviations. *Section I—Topical Remedies.* Explanatory notes. Medicated baths (No. 1-No. 4). Soaps, shampoos, and other detergents (No. 5-No. 18 Soaks and wet dressings (No. 19-No. 23). Liquid emulsions and oils (No. 24-No. 28). Shake lotions, magmas, and suspensions (No. 29-No. 38). Solutions, lotions, and tinctures (No. 39-No. 58). Creams and Ointments (No. 59-No. 115). Pastes (No. 116-No. 121). Powders and plasters (No. 122-No. 127). Lozenges (No. 128-No. 129). *Section II—Systemic Therapy.* Explanatory notes. A—Medicaments for Oral Use. Acids and antacids (No. 130-No. 132). Analgesics (No. 133-No. 134). Antibiotics (No. 135-No. 138). Antihistaminics (No. 139-No. 142). Hematinics (No. 143-No. 144). Hormones (No. 145-No. 150). Heavy Metals (No. 151-No. 153). Salts (No. 154-No. 158). Sedatives and hypnotics (No. 159-No. 162). Sulfonamides and sulfones (No. 163-No. 166). Vitamins (No. 167-No. 176). Miscellaneous (No. 177-No. 185). Antibiotics (No. 186-No. 188). Antihistaminics (No. 189). Hormones (No. 190-No. 192). Heavy metals (No. 193-No. 196). Sympathomimetic drugs (No. 197-No. 199). Vitamins (No. 200-No. 201). Miscellaneous (No. 202-No. 205). *Section III—Articles for Clinic Use.* Local anesthetics (No. 206-No. 210). Biologicals (No. 211-No. 215). Cauterizing agents (No. 216-No. 221). Dressings and bandages (No. 222-No. 231). Miscellaneous (No. 232-No. 242). Emergency kit for anaphylactic reactions. *Section IV—Therapeutic Aids.* Cosmetic and treatment aids (No. 243-No. 251). Directions for long-term corticotropin and/or corticosteroid therapy. Printed instructions issuable to patients. For care of acne. For care of impetigo and other pyodermas. For care of scabies. For home care of scalp ringworm. For varicose veins. For wet dressings. Index.

The ever-increasing spate of proprietary and ethical products now flooding the market is proving a great strain on the memories of prescribing physicians. The Dermatologic Formulary is a very useful, compact little volume which allows us easily to find the most suitable preparations for different dermatoses and to discover the constituents thereof.

Under the name of each preparation we are given its contents, its action and uses, the indications for prescribing it, directions for its application and its contra-indications and side effects.

Many useful hints are given; for instance, stains on the nails from potassium permanganate may be removed by 3% hydrogen peroxide, and bath stains by 'hypo', cream of tartar or household vinegar. Two sunscreen lotions, greatly needed in South Africa, are described, one containing menthyl anthranilate and the other para-aminobenzoic acid. Another substance often required in our warm climate is a satisfactory anti-perspirant tincture; the author gives one containing aluminium chloride in alcohol.

The book is very comprehensive, but I think that more side effects should have been cited; for instance, erythema nodosum is not mentioned as a complication of iodide administration nor is pellagra described during isoniazid therapy.

Gold is still said to be helpful in lupus erythermatosus and arthropathic psoriasis, but I think that it should be omitted from all modern books on dermatology because it is far more dangerous and less effective than the anti-malarial drugs.

Towards the end of the book some very useful directions are given for the care of patients undergoing prolonged therapy with the steroid hormones; schedules of treatment for acne, impetigo, scabies, tinea capitis and varicose veins are also listed.

This book covers the whole spectrum of the dermatologic formulary, ancient and modern and will prove an invaluable asset, not only to the harassed general practitioner, but to the busy dermatologist who cannot be expected to carry in his head all the details of the multitude of substances he may wish to use in treating his patients.

The science of Dermatology no longer consists of 'a name and an ointment'.

J.J.W.

GENERAL PRACTICE IN THE WELFARE STATE

The Doctor's Signature. By Hamilton Johnston. Pp. 255. 13s. 6d. London: Victor Gollancz Ltd. 1957.

This is the sort of book that could be an autobiography of any one of a thousand doctors. Dr. Truscott the likeable, moderately respectable, moderately successful and moderately honest general practitioner describes life in general practice in the Welfare State.

The adventures that befall him are discussed with humour and modesty, all sorts of characters familiar to all of us are brought before us, Dr. Craddock, successful and portentous, Dr. Chalk, inextinguishable, elderly and cranky. Dr. Peebles and others can all be recognized among any doctor's acquaintances. The book is full of humour and clean fun. It is an ideal present for Christmas or a present to a doctor on a sick bed but not for the post-operative case. The explosive character of the laughter that is associated with such a book would probably not be very good for the immediate post-operative case. Highly recommended for any of the profession who is feeling a bit 'browned off'.

S.T.T.

CORRESPONDENCE : BRIEWERUBRIEK

INCIDENCE OF DIABETES MELLITUS IN THE BANTU

To the Editor: In his letter[1] on this subject published in the *Journal* on 14 December, Dr. Keeley questions whether diabetes in the Bantu is as uncommon as we had indicated.[2,3] We should be glad to comment on the points raised in his letter.

Dr. Keeley states that a lower rate of diabetes would be predictable in the Bantu because of their lesser life expectancy. That is certainly true. In the United States, various reports suggest that from a quarter to a third or more cases of diabetes occur before 45 years.[4] Now in that country, for example in New York in 1950, 32·6% of the population were over 45 years and 7·7% were over 65 years;[4] among Bantu in Johannesburg, however, in 1954 the corresponding figures were 14 and 2%, respectively.[5] Hence, were the disease equally common among American and Bantu age groups, the incidence rate in the latter, taking into account the lower expectancy, should be only slightly more than half of that among Americans.

Referring firstly to hospital admissions: In a typical USA New England town, it was reported that 1·7% of admissions to hospital per annum were for diabetes.[7] If, on the basis of the foregoing, one uses half that figure, i.e. 0·85% for the Bantu, then in Baragwanath Hospital, one would expect 306 diabetic patients to be admitted from the 37,786 total admissions per annum in 1956. Dr. Keeley's figure for that year was 153. However, there are so many uncertainties latent in comparing causes of admission to Bantu and American hospitals that we cannot draw any inference from these particular figures.

Next, consider incidence rates: In the USA, for the whole population the figure has been given as 1-2%.[6] Taking into account the difference in life expectancy noted above, then for the disease to be equally common among the Bantu, the incidence figure in Johannesburg should be 0·5-1·0%, say 0·75%. Among the 539,000 Bantu in Johannesburg, in 1956 there should therefore be 4,043 diabetics. Dr. Keeley gives 153 diabetics admitted to hospital, and under 100 treated as out-patients. Clearly, we do not know the precise proportion of Johannesburg Bantu suffering from this disease who actually seek attention at hospital; nevertheless, the above data strongly suggest that diabetes is much less common in the Bantu compared with White populations.

Concerning younger populations, in the USA in 1945, in a study at Boston Induction Centre, among 69,088 male selectees for National Service aged 18-37 years, the incidence of diabetes was found to be unexpectedly high, namely, 1·1%.[9] In South Africa, among short-term Bantu mine-workers aged 18-40 years in one mining group—the Simmer and Jack Mines employing 7,500 workers—2 cases of diabetes were detected during the last 2 years, giving an incidence rate of 1/85 compared with the White group.[10] Of course, not all workers were tested for glycosuria, otherwise the figure might well have been higher; but undoubtedly, it would still be much lower than that of the American group cited.

It must be borne in mind, of course, that the Johannesburg Bantu (excluding the mine-workers) are to a varying measure westernized in food habit. In other countries it has been noted that sophistication of diet is associated with an increasing incidence of diabetes, and, incidentally, *vice versa*. Satisfactory information on incidence rates among *rural* Bantu are lacking, but such information as is available, combined with that given in annual and other reports from various territories in Central and Southern Africa, testify to the probably much lower incidence of diabetes among such people.

In dealing with diseases of this type, it is plausible to believe that many diseases formerly thought to be uncommon among under-privileged population groups require only careful search to reveal a much higher incidence. Taking into account Joslin's view of the 'universality of diabetes,[11] there is no doubt that the above belief is valid for this disease. At the same time, it must be emphasized that there *are* some population groups among which the disease is *very rare*. Joslin[11] admits this to be the case with the Navajo Indians (whom one of us recently visited), among whom Salisbury[12] was able to discover only 5 diabetics among 25,000 admitted to an Arizona mission hospital.

Dr. Keeley finally states: 'The influence of Bantu siderosis on the incidence of diabetes is as yet unknown; it might be expected to operate as an aggravating factor, becoming increasingly important the longer the individual lives.' In regard to the first part of the statement, we agree; regarding the expectation that siderosis may be an aggravational factor, we cannot agree. In the numerous studies on experimental animals with orally or parentally induced iron 'overload', pancreatic dysfunction has not been reported. Taking into account the conclusions reached in various recent reviews baring on siderosis in humans,[13–16] we very much doubt whether siderosis in the Bantu, directly or indirectly, is promotive of diabetes. Furthermore, may we point out that in Bantu siderosis, the pancreas is one of the organs least affected by increase in iron concentration.[17]

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I. Bersohn

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3 January1958

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CLYDESDALE MISSION HOSPITAL

To the Editor: I should be grateful if you would allow me to make known the needs of Clydesdale Mission Hospital (Anglican) in your *Journal*.

This hospital of 40 beds serves a thickly populated Coloured and African area in the District of Umzimkulu on the borders of Natal and the Transkei. The nearest hospitals are at Ixopo 16 miles on the Natal side and Kokstad 55 miles to the South. The demands of the area are for help in maternity and emergency surgery, as well as the full range of ordinary cases.

This small hospital needs a young and competent doctor with some experience, who would be prepared to take full charge. He will find himself confronted with hard work such as will make full demands on his resources physical, mental and spiritual. There is no doubt that he will find much to stimulate him, and that he will be given every opportunity for service.

The hospital also needs one or more nursing sisters of the same calibre as the doctor.

I am aware that I am asking a lot; but I am confident that there are young men and women to be found in this country ready to respond to such an appeal.

James S. Johns
Bishop of St. John's

P.O. Box 163
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23 December 1957